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FAREWELL TO "FORTY-FOUR".

By HARVEY SUTTON,

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ONE hundred years ago, the year 1844, saw the birth of a group of talented practitioners who, though they had not outstanding genius like Koch (1843), made real contributions to medical knowledge and skill and worked wisely for the welfare of their own and later generations.

Births in 1844.

Martin Bernhardt (1844-1915), of Berlin, with Roth described the Roth-Bernhardt disease *"meralgia paræsthetica"*.

Sir Thomas Lauder Brunton, baronet (1844-1916), came from Roxburghshire, qualified at Edinburgh, and after studying with Carl Ludwig at Leipzig, was appointed to the staff of Saint Bartholomew's Hospital, London. Following the inspiration of Ludwig, he was especially interested in the action of drugs on the circulation. His accidental sniffing of amyl nitrite, with the vigorous flushing and peripheral vaso-dilatation that followed, led him to work out its value in the relief of cardiac embarrassment, and especially of *angina pectoris*. Experimental work on animals and man elevated the study of drug action into a science, and Brunton wrote a leading text-book on pharmacology. The result of the scientific approach has been the discarding of many drugs in favour of few and far more effective remedies, and the clearer definition of the function of the chosen few. The condensation has been accompanied by an expansion, for the experimental basis has made possible the synthesis of more specific and less harmful new drugs. By destructive criticism and by constructive planning therapeutics has been rescued from the nihilism of the later nineteenth century on the one hand, and on the other from the narrowing confinement of systems, such as homœopathy, whose arrogant and at times fantastic assertions roused our grandfathers in the medical profession to transports of fury (see medical journals of the sixties in Australia). Brunton served on the Second Hyderabad Commission on Chloroform. Lauder Brunton was a man of far-seeing generosity and a true practitioner of the humanities. He devoted much of his later life to pressing the need for national preparedness, and was especially interested in school health activities. In recognition of his pioneering work he was made president of the International Congress of School Hygiene, 1907, and one of the editors of *The International Journal of School Health*.

G. Ceradini (1844-1894), a physiologist of Milan, wrote a history of the circulation (published 1906).

Francesco Durante (1844-1934), a surgeon of Rome, brought in the osteoplastic bone flap.

Camillo Golgi (1844-1926), of Cortona and later of Pavia, by use of his chrome-silver nitrate stain, helped to unravel the microscopic relationships of nerve cells and their branches. For this he, together with Ramon y Cajal, was awarded the Nobel Prize in 1908. Golgi showed the relation of fever to the parasitic life history in malaria.

Henri Huchard (1844-1910), of Paris, a leading clinician, studied especially arteriosclerosis and hypertension (Huchard's disease).

Victor Charles Hanot (1844-1896) described hypertrophic cirrhosis of the liver and primary endotheliomatous hypertrophy of the spleen.

Ludwig Hirt, of Breslau, born in 1844, wrote a monumental tome on occupational diseases (1871-1878).

Thomas Caverhill Jerden (1844-1916), of the Indian Medical Service, wrote on birds of India.

Sir Patrick Manson was born on October 3, 1844, at Old Meldrum, Aberdeenshire, and died on April 9, 1922, at London. After his graduation in Aberdeen in 1865 he went to Formosa attached to the Chinese Imperial Customs. Later he transferred to Amoy. Here, at the Mission Hospital in 1877, he carried out work on the life history and clinical importance of *Filaria* (the adult worm was found by Joseph Bancroft in Brisbane in 1876). Manson showed that part of the life cycle was spent in the Culex mosquito, and pointed out the periodicity of the filarial embryos. In 1883 he put up his plate in Hong-Kong and helped to inaugurate the medical school there for the Chinese. In 1890 he returned to London as a consultant. In 1894 he was convinced that the malarial parasite which Laveran had demonstrated was transmitted by the mosquito and inspired Ross in his successful attempt to prove this. The rectal parasite of schistosomiasis is named after him. In 1897 Manson evolved the idea of post-graduate instruction in tropical medicine to subserve the needs of a great and growing empire in the tropics. He convinced Joseph Chamberlain, that great colonial statesman, that all medical officers serving in tropical colonies should receive such training. London, Liverpool and Sydney are now recognized by the Colonial Office for this purpose. The Albert Dock Seamen's Hospital was the first place selected as receiving cases of tropical disease from all over the world. Although the Navy and the Army and Indian Medical Services for at least two centuries have made notable contributions to tropical research, university training came in with Manson, and this fact justifies his title of "father of tropical medicine".

Hermann Pagenstecher (1844-1908) and his brother built up at Wiesbaden an international reputation as ophthalmologists. Linen threads dipped in celluloid for use as sutures bear Hermann Pagenstecher's name.

Henri Parinaud (1844-1905), of Paris, reported on infectious tuberculous conjunctivitis (leptothrix) in 1899, and on the lachrymal pneumococcal conjunctivitis of the newborn.

Emil Ponfick (1844-1913) proved in 1880 the identity of actinomycosis in animal and man. He described shadow corpuscles.

Theodor Puschmann (1844-1899), of Vienna, a leading medical historian ("History of Medical Education", 1889), worked with others in the writing of Neuburger's great history.

Fulgence Raymond (1844-1910) succeeded Charcot at the Salpétrière, Paris, and wrote on *pseudotabes, progeria et cetera.*

Giuseppe Ruggi (1844-1905), an early abdominal surgeon, reported 1,000 successful laparotomies in 1886. Like Royle, he was interested in ramisection for spastic conditions.

Ernst Salkowski (1844-1923), of Berlin, investigated many biochemical problems in digestion and in the blood and urine, such as pentosuria, peptonuria, oxaluria. He wrote an important text-book for laboratory work in pathological chemistry.

Gustave Schwalbe (1844-1916) described the taste buds, *calyculi gustatorii.* He also wrote on the medullary nerve sheath and neurinoma. He also described the lymph spaces of the *lamina fusca* of the eye.

Nicolas Senn (1844-1909), Swiss by birth, practised and taught in Chicago. He visited Australia about 1902. His chief fields were military surgery and abdominal operations. Senn's decalcified bone plates for intestinal union preceded the Murphy button. He wrote on air embolism, the pancreas, appendicitis and leuchæmia. To find the perforation in the intestine he injected hydrogen *per rectum* and ignited the escaping gas with a match.

Lewis Atterbury Stimson (1844-1917), of New Jersey, was an authority and author on surgical technique, notably in fractures and in gynæcology.

The name of Friedrich Trendelenburg (1844-1924), of Berlin, adorns many surgical techniques, for example: Trendelenburg's sign in congenital hip dislocation, Trendelenburg's test and operation in varicose veins, Trendelenburg's position for pelvic operation (1890), Trendelenburg's tampon in tracheotomy.

Emile Troissier (1844-1919), of Paris, noted the enlargement of glands in the left supraclavicular fossa in late gastric carcinoma (lesser curvature).

A group of clinicians born in 1844 are known as the authors of various clinical signs *et cetera.* Emmanuel Aufrecht is known for Aufrecht's disease (infectious jaundice with nephritis) and for Aufrecht's sign in mitral stenosis (diminished sounds). H. M. Bannister, of Chicago (1844-1920), is remembered for Bannister's disease (angioneurotic œdema). Edoardo Bassini (1844-1924) is known for his radical cure of hernia. James Read Chadwick (died in 1905), an American gynæcologist, recorded the early sign in pregnancy of vaginal discoloration. Jean Henri D'Espine (died in 1931), a Swiss physician, discovered the echoed whisper over the first dorsal vertebra in mediastinal tuberculosis. Mathias Duval (died in 1915), a French anatomist, gave his name to Duval's nucleus near the hypoglossal nucleus. Heinrich Fritsch (died in 1915), a German gynæcologist, is remembered for the Bozemann and Fritsch catheter. Robert Gersuny, a surgeon of Bohemia, introduced paraffin injections for prosthesis. Alexis Joffroy (1844-1908) is remembered for Joffroy's arithmetical test in early general paralysis of the insane and for Joffroy's sign in exophthalmic goitre (absence of wrinkling of the forehead when looking up with head down). Max Marckwald, a German surgeon, devised an operation for stenosis of the *os uteri.* Sir Henry Morris (died in 1926), a surgeon of Middlesex Hospital, London, discovered the sign of remote bruising of the groin in renal injury associated with fractured pelvis (Bailey). Michel Julien Masselon, a physician of Paris, devised spectacles for ptosis and a test for mental disorder (sentence formation with three nouns). Alexander Ogston (died in 1929), a Scottish surgeon, devised an operation for knock-knee and its guide line, also an operation for flat-foot and one for frontal sinus disorder. He discovered the *Staphylococcus pyogenes aureus* as a cause of suppuration. He also wrote on his experience in three wars. Joseph Louis Renaut (died in 1917) investigated the basement membrane between the epidermis and the dermis (Ranvier's membrane). Henri Rendu (died in 1902) observed the intention tremor in hysteria, and also devised the treatment with camphor naphthol of tuberculosis of the peritoneum after laparotomy. Max Schede (died in 1902), a German surgeon, invented the use of organizing blood clot to fill the cavity left after removal of a sequestrum. Heinrich L. Schoeler devised the treatment of retinal detachment by the injection of iodine. Steinthal gave the first complete description of *tabes dorsalis.* Octave Terrillon, a French surgeon, suggested the use of an elastic ligature for the gradual removal of hydatid cysts.

One final name should be added, that of Robert Bridges (1844-1930), for, though he made his name in other fields, he was a distinguished physician. Bridges certainly came out of the "top drawer"—Eton, Oxford and Saint Bartholomew's Hospital—and his early education suggested a streamlined career as a consultant. His criticism of hospital management interrupted his progress at Saint Bartholomew's Hospital. After work on the staff of Great Ormond Street Hospital for Children he left medicine at the age of twenty-seven years to follow his first love—poetry and literature. In 1913 he became Poet Laureate. Probably few doctors are familiar with his poetry, with its intellectual view of life. As a master of good English he will figure in the future as one of the great writers of the early twentieth century. He was a well-built, fine-looking man, charming and sincere, and with a blind spot for publicity.

Happenings in 1844.

Among the technical achievements during the year 1844 may be noted the following: Washington Atlee, of the United States of America, performed the first successful myomectomy. Atlee performed 387 ovariotomies between 1844 and 1878. Agostino Bassi (1773-1857), of Lodi, who studied medicine under Scarpa, Spallanzani and Rasori, was the first to prove (1833) by experiment that living parasites (microscopic cryptogamic fungi) produced contagious disease (muscardine or calcino) in the silkworm. In a paper (1844) on the analogy of the silkworm disease

he urged that contagious diseases—smallpox, spotted fever, bubonic plague and syphilis—are produced by living parasites either animal or vegetable which pass from one individual to another. He advocated hospitalization of the sick and concurrent disinfection of eating utensils with boiling water and the bed linen and room with chlorinated water. He was indeed the precursor of Pasteur and Koch. Jean Boussingault, of Alsace, described the animal as an oxidizing and the plant as a reducing apparatus. He also pointed out the nitrogen cycle. Bunsen (1811-1899) brought out his grease spot photometer. He is perhaps best known by the Bunsen burner and by his work on spectrum analysis. Cahours found oil of wintergreen to be methyl salicylate and made it by synthesis. Charles Henry Ehrmann (1792-1878) first removed a laryngeal polypus. James Florian Heller (1813-1871) brought in the nitric acid ring test for albumin in the urine and used caustic potash as a test for sugar in the urine. Jacob Henle (1809-1885) first described casts in the urine. Friedrich Jolly noted the cases in which reaction of the muscle to faradic current is lost while voluntary action is retained. Carl Ludwig (1816-1895), of Marburg, outlined his theory of urinary secretion as a filtration action in response to blood pressure. Firmin Marbeau, on November 11, 1844, founded crêches as day nurseries for the infants of working mothers. Mitscherlich observed the rotatory property of tartrate crystals; this led to Pasteur's first piece of research. Josiah Charles Nott (1804-1873), of South Carolina, excised the coccyx for the condition later called coccigodynia. He is best remembered for his thesis that yellow fever is spread by mosquitoes as vectors. Max Pettenkofer (1818-1901), of Bavaria, while a pupil of Liebig, brought out his test for bile acids; later he estimated the carbon dioxide in atmospheric air. He became the first professor of hygiene (Munich, 1853). Plattner crystallized "bile" (sodium salts of bile acids). Robert Remak described the intrinsic ganglia of the heart as an argument for the neurogenic theory of its control. Carl Rokitansky (1804-1878) first recognized the essentially tuberculous character of Pott's type of spine. Edouard Séguin received from the French Academy of Sciences approval of his method of teaching the feeble-minded. James Thompson left England to establish a hospital in Damascus. Gabriel Valentin recognized the action of pancreatic juice on starch. He had already (with Purkinje) studied ciliary action.

Perhaps the most striking event of the year was the use of nitrous oxide as a dental anæsthetic agent by Horace Wells (1815-1848). He had a tooth extracted by Rigg when under its influence. He told his partner Morton, who later made a success of ether. Wells had a death with ether and took his own life.

Andrew Fyfe became the last mediciner (lecturer in chemistry) at King's College, Aberdeen, a post dating back to 1522.

In 1844 the Ethnological Society of London was founded and the Royal College of Veterinary Surgery received its charter.

The Health of Towns Commission sent in its report in 1844, and Ashley, Lord Shaftesbury, was the spearhead for reform.

Certifying factory surgeons were first appointed in 1844.

In Sydney in 1844 the Medico-Chirurgical Association of Australia was formed on the motion of Dr. McKellar "for mutual cooperation on all subjects connected with the profession". Dr. Bland was elected president, with George Bennet as secretary.

Important books published in 1844 include the following: Louis Agassiz (1807-1873), Swiss and American, the great comparative anatomist, completed his great work on fossil fishes. Golding Bird (1814-1854), of Norfolk, published his work on urinary deposits. The work of Bourdon on public hygiene was published. Thomas Caverhill Jerden (1844-1864) published "Birds of India". Daniel Drake (1785-1852), of Cincinnati, wrote on mesmerism. "Vestiges of the Natural History of Creation", published anonymously and written by Robert Chambers, anticipated in part "The Origin of Species". Charles Darwin's work on volcanic islands appeared. In 1844 Darwin wrote down his basic ideas on evolution in an unpublished essay of 231 pages. Henry H. Goodeve (1807-1884), professor of medicine at Calcutta, wrote "Hints on Children in India". Grisolle's work on "*Pathologie interne*" appeared. William Augustus Guy (1810-1885) wrote on legal medicine, and Taylor's work also appeared—the first books on the subject in English. Lecoq's work on ecology appeared. Justus von Liebig wrote "Familiar Letters on Chemistry". Alexander Jardine Lizars, of Aberdeen, wrote "Elements of Anatomy". Samuel George Morton (1799-1851), of Philadelphia, produced "*Crania Egyptiaca*", an illustrated atlas. Auguste Nélaton (1807-1873) wrote on surgical pathology. Nélaton's line is his best-known discovery—the tip of the trochanter touching the line from the anterior superior iliac spine to the *tuber ischii* when the hip is semiflexed. Filippo Pacini (1812-1823) wrote on the retina; as a student he had seen the Pacinian corpuscles in the skin, and in 1854 he described the vibrios of cholera. T. J. Pettigrew wrote on superstitions ("History and Practice of Medicine and Surgery"). Salvatore de Renza, who wrote on the Salernitan manuscripts, brought out a "History of Italian Medicine", 1844. D. B. Reed wrote on ventilation, and Selini wrote on the colloid state. The Sydenham Society produced the work of Louis on phthisis. Francis Gibson wrote "On the Position of the Internal Organs". His name is linked to the aorta and to the aponeurosis doming the pleura. Russell and Thacher Trall in the United States of America wrote on hydrotherapy. Gabriel Gustav Valentin wrote on physiology. Gustav Zimmermann (1817-1866) published work on newgrowths that disappear spontaneously ("*Pseudoplastische*").

Deaths in 1844.

The following died in 1844.

John Abercrombie (1780-1844), of Aberdeen, Edinburgh (M.D., 1803) and Saint George's Hospital, London, trained a series of apprentices, as many as five in one year, and built up at the Royal Public Hospital virtually a modern polyclinic. He is stated to have been the first consulting physician in Edinburgh and a clinical pathologist before clinical pathology became a university subject. He did much to systematize knowledge on the spinal marrow, the brain, the intestines and the heart. His last book was on "the culture and discipline of the mind". He described amyloid degeneration. Elected in 1835 as Lord Rector of Marischal College, he died in 1844 of rupture of a coronary artery. His brain weighed 64 ounces.

John Dalton (1766-1844), born at Eaglesfield, near Cockermouth, Cumberland, was one of those geniuses whom the race throws up from obscure origins to world fame. Son of a Quaker hand-loom worker, he made a scanty living as a school teacher and later as a private coach and at times as a public lecturer. At the school which he and his brother started, the boys preferred John; his mind was so occupied with mathematics that their faults escaped his notice. One of the periodicals of the day, *The Ladies' and Gentlemen's Diary and Woman's Almanac*, gave prizes to the person solving the most problems, mainly mathematical. In 1787 Dalton won with 13 out of 15. Even in questions more resembling those associated with "Dorothy Dix", John Dalton still gained the prize. His first love in science was meteorology, in which he recorded observations from 1787 to the very eve of his death in 1844. He performed early experiments on metabolism, weighing food and fluid taken and all ejecta to estimate the loss by insensible perspiration. At one time he thought of studying for medicine. He did, however, lecture to medical students in Manchester on chemistry, and was tutor in mathematics and natural philosophy in the New College. He also acted as a consultant analyst. Dalton dressed as a Quaker—knee breeches, grey stockings and buckled shoes, his suit of good broadcloth with spotless white neck cloth. He carried a handsome gold-tipped walking cane. A pipe smoker, he was very abstemious as regards alcohol; he found, like Helmholtz, that even small amounts drove away any chance of his arriving at any worthwhile scientific idea. Nevertheless he was once nearly poisoned by porter in which his analysis showed lead. He treated himself for colds by a mixture of liquorice, treacle and vinegar, with or without paregoric. He was an excellent walker and

robust mountaineer. His head was large and brachycephalic, the brain weighing three and a quarter pounds. Ransome, the surgeon, records that his features strongly resembled those of a death mask of Newton.

On October 31, 1794, Dalton read a paper on "Extraordinary Facts Relating to the Vision of Colours" before the Literary and Philosophical Society of Manchester, in which he first identified colour-blindness (long called Daltonism) in the persons of his brother and himself. He first realized the defect when in 1792, desiring to give his mother a birthday present, he bought her a pair of silk stockings, as he thought, of a dark bluish drab, a very proper sort of go-to-meeting colour. His mother was quite taken aback and asked what made him fancy such a bright colour: "They're as red as a cherry, John." He and his brother both thought her sight strangely out of order, but the neighbours confirmed her view of the stockings as "uncommon scarlety". While it seems strange today that an acute observer, naturalist and chemist, should live to the age of twenty-six years before realizing his colour defect, it is worth noting that it had never been discovered before. Still stranger is it, however, that about seventeen years before a Captain Huddart had written to Priestley of the abnormal eyesight of a family called Harris. Though the letter was published, it attracted no attention. Dalton followed this up and found that the Harris brothers were colour-blind. Dalton, checking various people against the spectrum, found that he agreed with their blue and purple, but that his yellow covered their red, orange, yellow and green. He records that he used to call pink sky-blue and people laughed, thinking he was merely joking. Tea and ale, "which others call brown I call green". Green cloth he thought like a mixture of mud two parts and red one part, grass and red sealing wax about the same colour, and the colour of a florid complexion to be dusky blue. No doubt, when Oxford conferred on him the degree of doctor of civil law, he accepted his scarlet gown as a good match for the green of the gardens and his own drab suit. Prévost, of Geneva, gave the name "Daltonism" to the defect, Wilson (1857) called it "chromatopseudopsis", Herschel called it "dichromic vision", but Brewster's "colour blindness" (1844) seems to be the usual description.

Dalton's chemical atomic theory he used to explain the facts of chemical combination. He estimated the relative weights of various elements. He first reported (1801) the equal expansion of all gases for equal increments of temperature (Charles's law). He extended Henry's law to the law of partial pressures and stated the chemical law of multiple proportions. The collection of Dalton relics, the proud possession of Manchester, was destroyed by a bomb in 1940.

Sir Henry Halford (1766-1844), Middlesex Hospital, was royal physician to four sovereigns. He was something more than a fashionable practitioner and did much for public health, presiding over the medical consultative committee called into action when cholera came to England. He rose to be President of the Royal College of Physicians. Once a year he took a month's holiday. What was then thought an eccentric habit now has become an almost universal practice. His name was originally Vaughan; the change occurred in regard to an inheritance.

Thomas Charles Hope (1766-1844) succeeded Black in the chair of chemistry and medicine at Edinburgh, and at one time had 500 students. He taught for nearly fifty years and was a master of "experimental demonstration". He is the discoverer of strontia (1798) and of the fact that water reaches its maximum density at 39·5° F. (4° C.), thus explaining why ice formed on the top of lakes and water pipes burst during frosts.

William Hey (1772-1844), a surgeon of Leeds, described (see Bailey's "Physical Diagnosis") a sign of unilateral dislocation of the mandible. He was the second Hey in a succession of three. He also wrote on "Internal Derangement of the Knee Joint" and invented Hey's saw.

Karl Kluge (1782-1844) induced premature labour by dilating the cervix with specially prepared sponges.

John C. Otto (1774-1844), a graduate of Princeton University, succeeded Rush at the Pennsylvania Hospital to take charge in the yellow fever epidemic of 1832 in Philadelphia. In 1803 he met with a family with a hæmorrhagic idiosyncrasy, later named by Schönlein "hæmophilia". Otto noted that many, but not all, of the male members were "bleeders" and that the female members were exempt, yet handed on the defect to certain of their sons. The usual local and general measures he found to be uniformly failures. He suggested purgative doses of sodium sulphate solution.

Etienne Geoffroy St. Hilaire (1772-1844), a follower of Lamarck, worked at embryology and evolution.

Births in 1744.

Edward Bartholomew Bancroft, who was born at Massachusetts in 1744 and died at Margate in 1821, ran away to sea as a lad. He probably gained medical experience as a surgeon's mate and practised on a plantation in Dutch Guiana. When twenty-five, he wrote on the natural history of Guiana. He described woorara (curare) "a preparation capable of perpetrating the most secret and fatal villainy". Coming to England in 1766, he was a student under Pitcairn at Saint Bartholomew's, taking his M.D. degree at Aberdeen. After publishing a novel in 1770, Bancroft began the long and successful study of colours and vegetable dyes and their production which won him his F.R.S. in 1773. His isolation of quercitrin from yellow oak made a "hit". He came from a distinguished family; he left distinguished descendants. He had many distinguished friends: Franklin and Paul Wentworth in America, Pringle, Priestley and Lettsom in London. He died universally honoured and respected, yet McNalty tells us that seventy years later documentary evidence showed that he combined the role of American patriot with that of secret British agent, and must be looked on as an unrivalled "double crosser".

J. Coquereau (1744-1796) was joint author of a work on elephantiasis, "*Le mal rouge de Cayenne*".

Pierre Joseph Desault (1744-1795) founded in Paris the *Journal de Chirurgie* (1792). Bichat was his pupil. Desault ligated the femoral artery at the adductor muscle to relieve popliteal aneurysm, to which the postilions at that time were prone. His name is attached to Desault's sign of rotation of the trochanter in fracture of the femoral neck, while Desault's splint for fractured femur, and Desault's bandage for fractured clavicle, levering the shoulder up and out over an axillary pad as fulcrum, are still in use.

Andrew Duncan, senior (1744-1828), Saint Andrews and Edinburgh, became professor of the Institutes of Medicine, 1789. He founded a number of societies, convivial, sporting and clinical. He obtained royal charters for the Royal Public Dispensary, which he founded, and the Royal Edinburgh Asylum at Morningside, 1813, for which he had actively agitated, also for the Medical Society and the Horticultural Society. He had many protégés. "His grave in Buccleuch burial ground is surrounded by small stones he erected to the memory of various students who died under his care and to whose remains he had accorded this posthumous hospitality." He died at the age of eighty-four years, "an aged, most amiable and benevolent believer in the good old times" (Currie).

Christian Gottfried Grüner (1744-1815) wrote chiefly on syphilis and the sweating sickness. In 1787 he showed that venereal infection could be transferred by a common drinking cup.

Jean Baptiste Pierre Antoine de Monet, Chevalier de Lamarck (1744-1829), had a most varied, versatile and brilliant career, being in turn theological student, soldier, physician, botanist and zoologist. His "Natural History of the Invertebrates" appeared between 1815 and 1822, and his greatest work, "*Philosophic zoologique*", in 1809. He is one of the foremost pioneers of evolution. His idea that variations arose from modifications of organs brought about by use or disuse, and that these acquired characters may be transmitted to offspring, is disputed. He thought, too, that structure followed function—a subject on which F. Wood Jones has written.

John Coakley Lettsom, M.D., F.R.S. (born November 22, 1744, died November 1, 1815), is of special interest to us as the first Dominions-born physician to rise to the top of the profession in London. He was born one of twins. His

was the seventh set, all males, and the only pair to survive. His father was a planter at Tortola in the Virgin Island, one of the Leeward group of the West Indies. At the age of six years he was sent off to school in England. As a Quaker he was taken up by the great Fothergill, a staunch member of the Society of Friends, and succeeded him as London's leading practitioner, at any rate as far as size of practice and degree of public spirit and generosity were concerned. He signed his prescriptions "I. Lettsom", and his memory has been and probably will be kept green by the well-known quatrain, of whose many versions Johnston Abraham—who has written on Lettsom, his life, times and friends, the best account of the medical world in the England of the later eighteenth century—selects the following:

> When any sick to me apply,
> I physics, bleeds and sweats 'em,
> If after that they choose to die
> Why verily!
>
> I. LETTSOM.

He helped to found the Medical Society of London and the Royal Humane Society, also the Prisoners' Aid Society. To him especially is due the credit for creating the Royal Sea Bathing Hospital at Margate. This is the genesis of the modern sanatorium. He wrote an interesting history of medicine and was the author of many papers on hygiene—stuffy air, bread substitutes, tea drinking, chlorosis in boarding schools, and the dangers of addiction to alcohol (1789). This is the first discussion of the drug habit. He wrote many articles for *The Gentleman's Magazine*, and may be regarded as the lineal ancestor of "The Doctor Who Tells".

Michele Vincenzo Malacarne (1744-1816), an Italian surgeon and pathologist, wrote in 1788 on cretinism and goitre. He described a lobule on the under surface of the vermis (Malacarne's pyramid), and the posterior perforated space (Malacarne's space).

Christian Ludwig Mursinna (1744-1823) rose from a barber's assitant to Surgeon-General of the Prussian Army.

Henry Park, a Liverpool surgeon (1744-1831), described arterio-venous aneurysm at the bend of the elbow.

Elisha Perkins (1744-1799—sometimes stated as 1740-1799), a reputable practitioner of Connecticut, probably deceived himself and certainly deceived thousands by his famous metallic or magnetic tractors. Made like a compass, the rod-like arms, one blunt, the other sharp-pointed, were built up of three metals, either copper, zinc and gold, or iron, silver and platinum. They cost five guineas a pair. Perkins held that if they were stroked down over the diseased organ the disease could be extracted. The new craze swept through the American colonies and later England. At the time Mesmer and his ideas on animal magnetism were one of the main topics for discussion in societies learned or otherwise. James Graham, of Aberdeen, had built in London his Temple of Health (1780), with an entrance fee of six guineas. Here "glamour girls" and soft music added atmosphere. Emma, Lady Hamilton, had been a priestess. His celestial beds at a fee of fifty pounds rejuvenated the senile and made fertile the childless. Suggestion was thus dramatized to the *n*th degree. Perkins's tractors received a knock-out blow when Haygarth showed that just as good results could be obtained with wooden dummies, while "Christopher Caustic" scarified the tractors in "Terrible Tractoration". Magnetic "cures" were first suggested by Paracelsus.

Happenings in 1744.

Among happenings in 1744 were the following.

Bishop Berkeley introduced tar water, a most curious episode. The practice of taking it became widespread, and it was unwittingly, no doubt, an early attempt at internal disinfection and a test of gustatory "guts".

Claude Nicholas Le Cat (1700-1768) published a work "On the Senses".

William Cullen (1710-1790), of Lanarkshire, began or virtually began the Glasgow Medical School in 1744. Later, at Edinburgh, he became the greatest teacher of his age, and the first to lecture in English. Many of the early surgeons who came to Australia must have been his students (see Monro).

Chlorine was evolved from dephlogisticated muriatic acid (hydrochloric acid).

Gerhard van Swieten (1700-1772) completed a lifetime study of cases, "*Constitutiones Epidemicæ*" (1727-1744). He was one of the first after Boerhaave to use the thermometer (Fahrenheit) and to administer perchloride of mercury in syphilis. Physician to the Empress Maria Theresa, he began clinical instruction in Vienna.

Alexander Monro published in 1744 "A Handbook of Comparative Anatomy". The three generations of surgeon anatomists, Alexander Monro, *primus*, *secundus* and *tertius*, held the chair of anatomy in Edinburgh from 1720 to 1846. Monro *primus* and Monro *secundus* taught 12,800 students between 1720 and 1790. This great triumvirate greatly helped to make Edinburgh the centre of medical education of the eighteenth century and after.

Frederick the Great found surgeons so scarce that in 1744 he authorized the performance of surgical operations by public executioners.

In 1744 Percival Pott started his long association with Saint Bartholomew's Hospital. In 1779 he wrote his classic paper on spinal deformity, first described by Platner in 1744.

William Smellie (1697-1763) introduced the steel lock forceps into obstetrics in 1744 and later (1751-1753) the curved and double curved forceps. His "Midwifery" (1752) first laid down rules for the diagnosis of contracted pelvis and the use of the forceps.

Abraham Trembley, of Geneva, a tutor, watching with a hand lens the life in the garden pond, saw a minute stalk with tentacles moving about and attaching itself to the green weed. When he cut it into two, tentacles grew, so that after eleven days he had two complete creatures. He wrote an exhaustive study (1744) on this fresh-water polypus (*Hydra*), proving it to have life without head, nerves, muscles or blood, and yet to belong to the animal kingdom and to be able to feed, grow, move about and find its prey.

Deaths in 1744.

Johann Heinrich Eder (1687-1744) wrote a history of medicine, and is said to have been the first to attempt photography.

Births in 1644.

Michael Etmuller (1644-1683) was one of the early iatrochemists.

William Salmon (1644-1713) wrote "The Family Dictionary", an early household book on medicine, "sold by Clavel at the Peacock, 1696".

Deaths in 1644.

Jean Baptiste Van Helmont (1577-1644) was born at Brussels. His father died when he was three years old, and his mother looked after his education and sent him, at the age of seventeen, to the University of Louvain. Arts, systematic botany, law, all alike failed to interest him; but medical studies, including Vesalian anatomy, apparently gave him what he sought. In 1599 he graduated, but he refused any academic distinction as ministering to pride. He contracted itch, and after suffering from the purging and ineffective internal dosing of the regular physicians, he was quickly relieved by a wandering quack by means of a sulphur preparation. This finished Galenical studies for him, and he switched over to the study of Paracelsus. Paracelsus first used alchemy as a source for chemical substances to use in treatment of disease. For long after the main source of drugs was herbs, but Paracelsus brought in chemical extracts and tinctures. As Francis Bacon summed up the position:

> Alchemy may be compared to the man who told his sons that he had left them gold buried somewhere in his vineyard; while they by digging found no gold, but by turning up the mould about the roots of the vines procured a plentiful vintage. So the search and endeavours to make gold have brought many inventions to light.

In Van Helmont we see the change over from the mystical. He is the great forerunner of scientific chemistry and carried out definite chemical experiments. The body processes were controlled by an archœus, the blas. The processes, he thought, were chemical and due to a special ferment or gas. Indeed, he created the word gas, probably from the word chaos. This is recorded in his journal, "*Hunc spiritum, hactenus ignotum, novo nomine gas voco*".

Van Helmont studied yeast and fermentation of beer and wine and observed the production of the gas sylvestre, carbon dioxide, also its origin from limestone. He broke away from the doctrine of the four elements and held that water was the one and only element, the essential principle in nature. He planted a young willow weighing five pounds in a pot holding 200 pounds of earth baked in a furnace. The plant was protected from dust, and water was freely supplied. After five years the tree had grown to 169 pounds, thirty-three times the original weight, while the weight of earth remained unchanged. In 1644 he worked out the preparation of copper sulphate, and he understood the reaction of metal and acid to form a salt. Of drugs he invented *laudanum cydoniatum* — opium dissolved by vinegar. The central tendon of the diaphragm has been called Van Helmont's mirror, *speculum Helmonthi*. In his "*De magnetica vulnerarum curatione*", 1644, he tells of the mysterious power of sympathy:

> A citizen of Brussels had had his nose cut off in a fight. Tagliacozzi made him another, taking a strip of skin and flesh from a servant. About a year later the nose felt cold and began to putrefy. The servant had died!

On the other hand, he gave a physical explanation for certain miracles, which brought him ecclesiastical displeasure and discipline; he was forced to live for some time under house arrest. Van Helmont lived and worked at Vilvorde, near Brussels. He placed the soul in the pit of the stomach, the "solar plexus". Osler thought he had some real idea of immunity. He had a good deal of knowledge about gastric juice and bile. He founded the iatrochemical school.

William Gascoigne (1612-1644) invented the micrometer for measuring small angular distances. Huygens introduced this into the telescope in 1658.

Thomas Johnson (1600-1644), apothecary and royalist colonel, killed at Basinghouse in the Civil War, published "Gerarde's Herbal" in 1636.

Happenings in 1644.

Descartes published his "Principles of Philosophy" and his "Dioptrics" in 1644. René Descartes (March, 1596, to February, 1650) grew up a delicate boy, thought to be doomed to an early death, as his mother had died of tuberculosis of the lungs. At boarding school he was allowed to sleep in and attend classes when he felt fit. The habit of sleeping in became a lifelong habit. From the age of sixteen to that of twenty-three years he was a private soldier, but he still had his twelve hours or more per day in bed. On the eve of November 11, 1619, conviction gripped him, linked with vivid dreams, that his life work must be the search after truth, and that mathematics would give the clue. He brushed aside book learning, "refusing to be deceived by the professions of the alchemist, the predictions of the astrologer, the impostures of the magician or the artifices of any of those who profess to know things of which they are ignorant". After years of wandering as a scholar at large, in 1629 he settled down in Holland. As a gentleman of leisure he tells us that "he was not compelled to make a merchandise of science for the betterment of my fortune"; yet, as an amateur, he wrote the first modern work on physiology and founded analytical geometry. In 1644 he published his "principles" (*Principiæ Philosophiæ*). This is an exposition of his philosophy, in which he puts forward his theory of vortices. He mentions reflex action in the response of the eyelids to a threatened blow. In "Dioptrics" he, like Snell, compared the eye to a camera. He realized that accommodation was the work of the lens.

Descartes's main physiological treatise is that on "man" (*De Homine*) and on the formation of the fœtus. He emphasized the mechanical aspect of human function—man as a kind of complicated clock. He looked for the seat of the soul in some single organ, centrally situated, deeply placed and fully protected, and so he "plumped" for the pineal gland. What a pity he did not give some attention to the pituitary!

John Bulwer wrote on deaf mutes, 1644-1648. Emperor Chen Lung edited an encyclopædia of medicine in China. Feng Chiao Chang wrote on regional anatomy. Isbrand Diemerbrock (1609-1674) published his studies of the plague in 1644. The Hôtel Dieu was founded at Montreal. This is the earliest hospital foundation in the Empire outside the British Isles, with the exception of the Hôtel Dieu at Quebec, which was founded five years before. The first hospital in the New World was founded in 1524 in Mexico by Cortes. In 1644 also Ruland published his pharmacopœia.

Births in 1544.

William Gilbert (or Gilberd) (1544-1603), of Colchester, Fellow of Saint John's College, Cambridge, graduated as doctor of medicine in 1569. After a tour of Italy he set up practice in London in 1573 as a Fellow of the Royal College of Physicians and physician-in-ordinary to Queen Elizabeth. At his house near Saint Paul's a group of philosophers held meetings, the first group of the kind in England. He is our first great scientist since Roger Bacon—over three centuries before. In 1600, when Gilbert was President of the Royal College of Physicians, he published his epoch-making work, "*De magnete magneticisque corporibus et de magno magnete tellure, physiologia nova*". He is the father of electromagnetic science. Fuller states that Queen Elizabeth "stamped on him many marks of her favour besides an annual pension to encourage his studies . . . He was stoicall but not cynicall . . . reserv'd but not morose; never married, purposely to be more beneficial to his brethren . . . His stature was tall, complexion cheerful, an happiness not ordinary in so hard a student and retired person. He lyeth buried in Trinity Church in Colchester under a plain monument. . . . Mahomet's Tomb of Mecca is said strangely to hang up attracted by some invisible loadestone but the memory of this doctor will never fall to the ground which his incomparable book '*De Magnete*' will support to Eternity."

In his preface to his wonderful book Gilbert stressed the need for verification by experiment and experience or, in other words, the scientific method. He objected to "the judgement of men who have taken oath to follow the opinions of others . . . to lettered clowns, grammatists, sophists, spoilers, and the wrongheaded rabble". In his book the picture is given of a smith beating a bar of iron placed in a north-south position, thus magnetizing it. The Greeks knew that after amber had been rubbed ("electron") it would pick up small pieces of paper. The lodestone was well known and thought to produce melancholy and to be useful in love philters. Its powers were thought to be activated by goats' blood and lost if it was rubbed with garlic. Gilbert showed that many other substances could be magnetized. He proved the earth itself to be a magnet, thus explaining the magnetic dip of the needle with its north-seeking and south-seeking ends. He was the first to advocate Copernican views in England, and held the fixed stars to be at varying distances from the earth. Two instruments to find latitude without seeing the sun, moon or stars are ascribed to him.

Deaths in 1544.

Valerius Cordus (1515-1544), of Erfurt, was the son of a physician and botanist. In spite of his early death, we owe to him the first real pharmacopœia published, the "Dispensatorium", 1535, Nurnberg, which went into 35 editions. Like so many doctors of the time and since, he was a keen botanist and described over 500 new plant species in an almost modern systematic fashion. He also wrote a commentary on Dioscorides, and introduced in 1540 sulphuric ether (*oleum dulce vitrioli*).

Happenings in 1544.

Guido Guidi (Vidius), who died in 1569, is well known to anatomists (Vidian nerve and canal). Though a Florentine, he became royal physician and professor at the *Collège de France*, but later returned to Pisa. He wrote in Latin a well-illustrated book on surgery

("*Chirurgia*", 1544), chiefly based on the Greeks, and described the pons and brain stem. He confirmed what Vesalius had hinted at—namely, the absence of pores through the septum.

William Turner (died 1568), a college friend of Ridley and Latimer, fled to the Continent to avoid religious persecution. There he studied botany and medicine. In 1544 his history of birds, followed by a herbal with many British plants (1551), shows him to be a pioneer English naturalist. Many of the birds then common—for example, the crane, the osprey and the kite—are now rare.

John Caius, a contemporary, who similarly wrote about British dogs (1570), lists a "turnspit" but no bulldog or pointer.

The University of Königsberg was established by Albert III in 1544.

Pietro Andrea Matteoli (1501-1577) published in 1544 a commentary on *Dioscorea*, adding over 200 new species from southern Europe. Dioscorides, who flourished in the reign of Nero, was the first to write on medical botany. His works remained the text-book of *materia medica* for 1,600 years.

In 1544 Pisa set up one of the earliest botanic gardens. These were largely devoted to herbs and drug plants.

Vicary in 1544 helped to reestablish the activities of Saint Bartholomew's Hospital, one of the three hospitals that survived the suppression of monasteries *et cetera* by Henry VIII.

The suppression of the religious houses by Henry VIII ended with one fell swoop the help they gave to the sick and needy. Public compassion, however, aroused by the sight of helpless blind and infirm on the streets, moved the mayor and aldermen of the City of London to petition the King. They asked "Our most redoubted puyssant and mighty prynce, our most drad beloved and naturall soveraign lorde" that the hospitals of Saint Mary's (of Bethlehem), of Saint Bartholomew and of Saint Thomas and the New Abbey Tower Hill might be placed under their control "for the ayde and comforte of the poore sykke blynde aged and impotent persons being not hable to helpe theymselffs nor havying any place certeyn whereyn they may be lodged cherysshed and refresshed tyll they be cured and holpen of theyre dyseases and syknesse . . . frankly and freely by physicions surgeons and appotycaryes which shall have stypende salary and wages onely to attende for that entente and purpose".

Five years later, on June 23, 1544, the King granted letters patent for Saint Bartholomew's Hospital to be restored and renewed. The new corporate body, however, seems to have been mainly religious in type and made up of court favourites, the King's chaplain being master. The grant did little for the poor, and not till 1547 was this righted. The hospital did in 1544 receive a portrait of King Henry VIII, then aged fifty-three years, which in normal times decorates the committee room of the hospital.

In 1544 Vesalius became court physician to Charles V and later to Philip II. Apparently his desire to dissect eventually led him into trouble in that intolerant society. What actually happened has never been made clear, but Vesalius, as a penance, visited the Holy Land and on the way back died at Zante Island (Zakynthos) in the Ionian Sea in 1564.

Happenings in Earlier Centuries.

In 1244 Oxford received a charter from Henry III. A riot broke out in which the clerks, in revenge for extortion, sacked the houses of the Jewish money-lenders. This was unusual, most of the riots being between Town and Gown. The great Bishop of Lincoln, Robert Grosseteste (Greathead), was chancellor (or master) of the scholars at Oxford. At Bishop Grosseteste's urgent demand the errant clerks were transferred from the civil to church custody. The King issued a decree that all debts and suchlike disputes should be decided by the chancellor should a clerk of Oxford be involved. Even clerks trespassing in the royal forest armed with bows and arrows were handed over to the chancellor. This may be justly regarded as the Magna Charta of the university. Roger Bacon greatly praised Grosseteste's knowledge of the sciences, and, like other Franciscans, he had diligently studied practical medicine.

In 1248 Henry III extended the charter, limited "interest" to 43% and gave to newly appointed proctors the right of being present at the assay of bread and ale.

In 1144 Robert of Chester (1110-1160) wrote the first alchemical text to be rendered into Latin. He had spent some time with the Moors in Spain and learnt Arabic there.

References to events occurring before the Christian era are both scanty and difficult to verify.

The Græco-Roman period may be conveniently dated as lasting from 156 B.C to A.D. 576, thus beginning 2,100 years ago. Its greatest medical genius was Galen.

Two early Greek scientists may be mentioned. The first is Eudoxus of Cnidus (409-356 B.C.), a famous medical centre and rival of Cos, who studied the heavens from a mathematical point of view, making an accurate estimate of the solar year. He was one of the first to construct a sun dial. He died 2,300 years ago. The second is the Ionian Heraclitus of Ephesus (556-460 B.C.), who was born 2,500 years ago. He was accounted one of the wise men of Greece and was sometimes called the weeping philosopher. Fire he regarded as the symbol of existence. Everything he thought was in a state of flux. Allbutt calls him "the loftiest and the most positive genius of all these early sages" and considers that he had a prevision of the conservation of energy. "Change and decay in all around" he saw, but "nothing of all things either perishes or is generated which was not in existence before". Fire, air, water, earth became the basic elements of Greek medicine.

DISEASE OF THE GALL-BLADDER.

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THE following statement is taken from Rehfuss and Nelson's book "Medical Treatment of Gall-Bladder Disease".[1] As the authors are respectively Clinical Professor of Medicine and Instructor of Medicine at the Jefferson Medical College, Philadelphia, they can be absolved from any prejudice in favour of surgery. The statement is as follows:

> We now realize that gall-bladder disease is the commonest upper abdominal trouble; 25% of the adult population have gall-stones, 25% have cholecystitis without stones; in fact 40% to 50% of the adult population have disorders of the biliary tract which may at any time give rise to active symptoms.

Disease of the gall-bladder is a vast subject, and my talk must be a mere skirmish. It is interesting to know that abdominal section was introduced by Alkmaion,[2] of Crotona, in 500 B.C., and that his pupil Philolaos[3] later propounded the theory that disease was due to four elements, black bile, yellow bile, blood and phlegm; but no mention has been found by medical historians of the occurrence of gall-stones till they were recorded by Traillanus[4] in A.D. 600. There was then a gap of over a thousand years, till Fabricius Hildanus[5] in 1618 removed a gall-stone from a living patient; then another lull to the time of Bobbs,[6] Keen,[7] Sims[8] and Tait,[9] who in 1879 laid the foundations of the modern operation for gall-stones which we know today.

Lawson Tait taught Mayo Robson, and he in turn Moynihan—all world figures in the realms of gall-bladder surgery. If any of those present are interested in the history of gall-bladder surgery they will find excellent articles dealing with the subject in Stewart McKay's[10] history of Lawson Tait and Moynihan's[11] book on gall-stones.

But the diseased gall-bladder is still a "problem child" and never ceases to cause surgical headaches.

Development of the Gall-Bladder.

The gall-bladder is developed as an outgrowth of the common bile duct, which in turn arises from the primitive foregut, having an epithelial lining and a mesodermic back-

ground, and is naturally subject to all afflictions common to similar structures in the human body.

Functions of the Gall-Bladder.

The function of the gall-bladder has been variously assessed by many writers, as follows: (i) as a reservoir of bile; (ii) as a safety valve taking tension from the common and hepatic ducts; (iii) as a concentrating chamber; (iv) as a regulator of biliary flow; (v) as an active secreting structure; (vi) as a vestigial remnant. We know that the gall-bladder is not present in some animals, such as the horse, the ass, the zebra and the rhinoceros, but is present in the cud-chewers, such as the ox, the sheep and the camel, and in pigs, dogs and cats. It is safe to assume, then, that the normal healthy gall-bladder has some important role to play in man.

Anatomy of the Gall-Bladder.

The fundus lies from ten to twelve centimetres but the neck only three and a half centimetres from the middle line, and the neck lies just above Addison's plane—that is, at the level of the first lumbar vertebra. The body of the gall-bladder is bound to the liver by intervening cellular tissue, and through this pass the lesser cystic vessels. Sometimes the body of the gall-bladder is suspended by a mesentery from the liver, and when this type is present one finds a "flopping" gall-bladder, which is easily kinked and often the seat of biliary stasis even simulating cholelithiasis. The cysto-colic ligament—that is, the outlying portion of the lesser omentum—is present in one out of four subjects and passes from the under surface of the gall-bladder to the second portion of the duodenum.

The neck of the gall-bladder forms an acute angle in a direction parallel with the body. The body, the neck and the cystic duct form a Z-shaped structure, and at each bend there is a reduplication of the mucous membrane forming a valve-like partition, which, with several other partial foldings of the mucous membrane of the gall-bladder, makes the passage of a probe from the gall-bladder into the common duct almost impossible. Diverticula are frequent at the junction of the body and neck, and one in particular is known as Hartmann's pouch and is often the starting point of stones.

The cystic duct is the narrowest part of the bile passages and passes up and to the left and then turns down and meets the hepatic duct. The cystic and hepatic ducts lie in the right free margin of the lesser omentum in front of the foramen of Winslow and join behind the first part of the duodenum to form the common duct. It is when the cystic duct is dilated after a recent attack of biliary colic, even though stones are present in the gall-bladder and there is no history of jaundice, that one must be on the alert for a pebble in the common duct.

The common bile duct, which is sometimes the seat of many abnormalities, consists of a retroduodenal, a pancreatic and an intramural portion. The duodenal part is in close relation with the inferior *vena cava*, the portal vein and the gastro-duodenal trunk of the hepatic artery, and according to many anatomists, it opens into the bowel by Vater's papilla by a single orifice in about 75% of persons. The common bile duct grows smaller in calibre as it reaches the duodenum, and thus stones are more frequently met with in this position.

The blood supply of the gall-bladder comes from two sources, from the cystic artery itself and from the lesser cystic arteries from the liver. The cystic artery usually arises either from the hepatic trunk or from the right branch of the hepatic trunk, and lies above and to the left of the cystic duct; but, like the common duct, it too is subject to many irregularities.

The cystic duct gets its blood supply from the cystic artery; the hepatic ducts and the upper part of the common bile duct receive their blood supply from the hepatic trunk, while the lower part of the common duct gets its blood supply from the right superior pancreatic-duodenal artery.

The portal vein lies to the lateral side of the upper third of the common duct and may overlap it in front, and is liable to injury in operations on this part of the common duct. As it is this part of the common duct which should be opened when a search for stones is being made, this relationship must ever be kept in mind.

The lymphatics of the gall-bladder go to the hilum of the liver and join the efferent vessels of the liver and connect with those from the common duct. They all unite in a plexus, and with those of the duodenum drain into nodes in the immediate neighbourhood of the head of the pancreas.

Experiments of Berceanu[12] have proved the existence of a direct lymphatic path from the appendix to glands in the duodeno-pancreatic region, and it would thus seem that the lymphatics are the chief connecting links between an evil triumvirate of the abdomen, the appendix, the gall-bladder and the pancreas. The duodenum is closely associated with these structures. Infection in any of them often leaves as a legacy intractable periduodenitis.

The main nerve supply to the gall-bladder (Mackenzie[13]) comes from the eighth and ninth dorsal nerves via the solar plexus, and according to Bainbridge and Dale's[14] experiments the vagus nerve gives some motor fibres. The phrenic nerve (the third, fourth and fifth cervical segments) is also considered to give a supply, and by these paths referred pain from the gall-bladder and duct is frequently found in the right lower scapular region (eighth dorsal segment) and in the right arm and right upper scapular region (third and fourth cervical segments).

Causes of Infection.

The gall-bladder is like any other organ of the human body, in that infection may be derived from any source, such as infected teeth, nasal sinuses, alimentary tract or genito-urinary system, and when once infection enters the blood it can obtain access to any part of the body, and particularly to the gall-bladder, especially if the liver has to act the part of the septic tank and cleanse the fluid passing through.

Infection may reach the gall-bladder by many routes: from the duodenum, by way of the common duct—and it is of interest to note that Butt[15] reports a case of round worm in the gall-bladder; from the liver through the portal circulation and thence by the bile; from the blood and lymphatics; or attack may come through adhesions of the gall-bladder with some neighbouring organ.

Much work has been carried out on the investigation of achlorhydria. Hurst,[16] in 762 consecutive cases, found achlorhydria in 15%, and Langdon Brown[17] states that 5% of the population are achlorhydric. In many of these people a streptococcus has been found in the duodenum, and thus achlorhydria and its attendant duodenal infection must also be considered as a possible cause of infection of the gall-bladder.

The belief amongst most investigators today is that infection of the gall-bladder is mainly blood-borne. Wilkie[18] proved this to his satisfaction after many years of observation and animal experimentation.

Research goes to show that gall-stones, apart from the pure cholesterol stones, are preceded by an infection, and in support of this Gross[19] in 9,531 autopsies found (i) 802 cases of gall-stones, (ii) a definite association between feceted pigmented stones and cholecystitis, and (iii) no cholecystitis existing with cholesterol stones. Walton[20] lays stress on the connexion between urinary and gall-bladder infections, pointing out that the two conditions are closely associated and are manifestations of chronic septicæmia. His opinion is that the more common occurrence of the *Bacillus coli communis* organism in pregnant women would as one factor help to explain the more frequent occurrence of gall-stones in women than in men (two to one), though, of course, constipation is more prevalent in women. Out of 347 women patients of Walton at the London Hospital who were afflicted with gall-stones, 324 had been pregnant.

Extensive and searching experiments by Rosenow[21] on animals by intravenous injections of bacteria with proven affinity for various abdominal organs—for example, the stomach, gall-bladder, duodenum and appendix—demonstrated that he was able in a great percentage of cases to produce inflammation in these organs typical of the sites from which the organisms were cultured, but he was unable to produce cholecystitis by injecting them directly into the

gall-bladder. The results of his experiments with numerous varieties of organisms showed the streptococcus to be the chief cause of ulcer of the stomach, cholecystitis, appendicitis and pancreatitis.

Observation on gall-bladders removed by several authorities have demonstrated sterile bile in the gall-bladder, and in the same gall-bladder wall numerous organisms have been found.

To sum up the pathology, what happens in most gall-bladder infections is that biliary stasis takes place, probably with an increased cholesterol content in the blood and bile associated with constipation, then infection of the portal blood occurs, resulting in cholecystitis.

Symptoms of Gall-Bladder Disease.

The symptoms of gall-bladder trouble are generally referred to the stomach. Most frequently a flatulent dyspepsia occurs in the form of discomfort half an hour after food, relieved by the belching of wind and more pronounced after certain foods, such as fat, pastry and vegetables. In other cases the gastric symptoms show themselves an hour or more after food, as heart-burn and acid eructations.

Pain is an important symptom in all cases of cholecystitis. It may vary from a little discomfort after food to severe pain referred to the right costal margin and round the lower right ribs to the lower angle of the scapula via the eighth dorsal nerve. In a small minority of cases the pain extends to the right iliac fossa, simulating appendicitis or renal trouble, and an important point to remember is that the three conditions may coexist.

On occasions pain on the left side is the more prominent, and when accompanied by palpitation suggests to the patient cardiac trouble—a condition spoken of by physicians as "cholecystic heart"; in fact *angina pectoris* may be closely simulated.

The accompanying tables (Tables IA and IB), drawn up by Faulkiner, Marble and White[22] show how certain aspects of the clinical pictures of coronary occlusion and attacks of cholelithiasis may simulate each other—in fact, may coexist. Note particularly the tenderness in the upper quadrant, the epigastric pain, the nausea and vomiting.

TABLE IA.
(*After* Faulkiner, Marble and White.)[1]

Position of Pain.	Coronary Occlusion.	Cholelithiasis.
Precordial	2	0
Substernal	2	0
Chest	1	0
Both precordial and epigastric	2	0
Epigastric	(5)	(15)
Lower part of abdomen	2	0
Diffuse over abdomen	1	0
Right upper quadrant	0	9
Right upper quadrant and epigastrium	0	1
Lower part of right side of thorax	0	2
Angle of right scapula	0	1
Left breast	0	1
Both flanks	0	1

TABLE IB.
(*After* Faulkiner, Marble and White.)[1]

Symptoms Associated with the Pain.	Coronary Occlusion.	Cholelithiasis.
Nausea	(5)	(12)
Vomiting	(7)	(16)
Palpitation	3	0
Dyspnœa	17	0
Syncope	6	0
Faintness	5	0
Vertigo	3	0
Weakness	4	2
Tenderness in right upper quadrant	(5)	(23)

[1] Thirty consecutive cases of occlusion of a coronary artery were found at post-mortem examination, and 30 consecutive cases of cholelithiasis were confirmed at operation.

In those cases in which the pain is referred through the fourth and fifth cervical nerves, neuritis is suggested from the aching in the right shoulder and the right side of the neck and down the right arm. Occasionally symptoms are referred to the right breast via the eighth dorsal nerve. Wilkie[23] mentions relief resulting from cholecystectomy in cases of chronic arthritis, toxic myocarditis, toxic neuritis, toxic hepatic cirrhosis and toxic mental derangement, and I can call to mind many similar cases in which relief resulted in the same way.

The above symptoms refer to the normally placed gall-bladder. In rare cases complete transposition of the abdominal and thoracic viscera is present, and several cases are on record of operations for acute cholecystitis on the left side.

The gall-bladder sometimes extends as far as the pelvis and simulates pelvic trouble. Bland Sutton[24] reports a case in which the gall-bladder was found in a right hernial sac, another in which he operated for a subserous fibroid tumour of the uterus but found a cancerous gall-bladder attached to the fundus of the uterus, and still another in which Lawson Tait operated for an ovarian cyst and found a huge mucocele of the gall-bladder.

Gall-bladder trouble may be overshadowed by symptoms of pancreatic infection, the result of the diseased state of the gall-bladder, and this can be easily understood from the experiments of Harer, Hargis and von Meter,[25] which revealed the intimate association between the lymphatics of the gall-bladder and the pancreas.

At the beginning of the discussion of symptoms it was stated that gastric upset was a prominent feature of gall-bladder trouble; Duvan, Roux and Béclère,[26] from a series of radiographic studies, were able to demonstrate that if periduodenitis exists before cholecystectomy it may persist after operation, and that the existence of gall-stones with severe gastric disturbance suggests an accompanying periduodenitis, which in some cases may be so severe as to cause duodenal stenosis from adhesions and suggests malignant obstruction.

Diagnosis.

It has to be remembered that the formation of calculi and attempts to expel them from the gall-bladder and ducts are terminal events in the history of cholecystitis. Prevention is possible apparently, for McCarrison,[27] after nine years' service in the Himalayas, during which period he averaged 400 major operations every year, did not see one case of appendicitis, one ulcer of the duodenum, one case of dyspepsia, colitis, cholecystitis or cancer.

There are many means at our command to help solve the riddle, but nothing is more helpful than an accurately and carefully compiled clinical history. Added to this, radiographic evidence is of great value, both direct and indirect—direct in that in a small percentage of cases shadows may be obtained of stones in the gall-bladder; indirect by pressure effects—for example, on the duodenum and stomach—or by traction on and thus displacement of the duodenum, or by fixation and thus displacement of the colon. George[28] many years ago laid particular stress on the pressure and traction effects on neighbouring viscera exerted by a diseased gall-bladder. In all cases an opaque meal should be given as well as the dye to exclude any œsophago-gastro-intestinal lesion. The cases of W.A. and G.S. emphasize this necessity.

W.A. was admitted to a public hospital on April 1, 1939. He had a history of attacks of pain in the epigastrium, right hypochondrium and right loin of eight weeks' duration, and of anorexia and nausea for three months before his admission to hospital. He suffered from flatulence, but no loss of weight. There was no history of jaundice or clay-coloured stools. The bowels were open regularly and the stools were well formed.

On examination, the patient was pale, healthy and thin. No jaundice was present, but tenderness in the right hypochondrium was elicited. X-ray examination on April 5 disclosed a normal stomach and duodenum. No report was made on the colon.

At an X-ray examination of the gall-bladder on April 14 the gall-bladder was not visualized. Operation on April 20 disclosed free fluid in the abdomen; the gall-bladder was removed, as well as a cancer of the colon, and colostomy of the double gun-barrel type was established. On May 3 the

spur of the colostomy was crushed; on May 17 the colostomy was closed; and on June 8 the patient was discharged from hospital with the wound healed and the bowels acting normally.

G.S., aged fifty-six years, was admitted to a public hospital on November 1, 1938, complaining of attacks of pain in the area of the gall-bladder and in the retrosternal and interscapular regions. Anorexia, nausea and flatulence were present, but no jaundice; the patient was a heavy drinker and smoker, suffered from breathlessness on exertion, and had lost two stone in weight in five months (from seventeen to fifteen stone); this he thought was due to dieting.

On examination, the patient was fat and flabby; the systolic blood pressure was 125 millimetres of mercury and the diastolic pressure 90. No abnormality was detected in the abdomen. X-ray examination of the gastro-intestinal tract revealed no abnormality. The condition of the œsophagus was not mentioned. X-ray investigation of the gall-bladder disclosed an abnormal condition.

Operation was carried out on November 23, 1938, and a strawberry gall-bladder with its walls infiltrated with fat was removed. The operation was considered unsatisfactory, as the abnormal condition of the gall-bladder did not seem to measure up to the symptoms. The patient was readmitted to hospital on January 16, 1939, on account of difficulty in swallowing. X-ray examination disclosed obstruction of the œsophagus. On January 25 gastrostomy was performed, and on February 19 the patient was discharged from hospital.

Graham's test is now thoroughly well established, though there is still a small margin of error.

Still another means devised for diagnosis and treatment is the Meltzer-Lyon test. This test consists of the instillation of a strong solution of magnesium sulphate into the duodenum by means of a tube, with the object of paralysing reflexly the sphincter of Oddi, to cause gall-bladder contraction and thus allow collection of bile from the common bile duct and gall-bladder and liver bile. A thorough examination of each specimen, both cultural and microscopic, is then made.

The Van den Bergh test is used to distinguish between obstructive and non-obstructive jaundice. Jaundice may be present, but as a rule it occurs only when infective cholangitis or indirect obstruction of the common duct by a stone is present, or when stones are present in the cystic duct of an acutely inflamed gall-bladder, or when the common duct itself is obstructed by a calculus or calculi. Too great stress is laid on the presence or otherwise of jaundice as a diagnostic sign of gall-bladder infections or calculus formation. A history of its presence is helpful, but its absence by no means rules out stones or infection.

Much work has been done on the jaundice problem and hepatic efficiency, but no definite test has yet been discovered to allow any accurate estimation of liver function. The age of the patient is of some help, in that stones do not as a rule occur before middle life; but in rare cases they are met with at a tender age. To meet them in people aged under thirty years is quite common, but often they do not make their presence felt till ripe old age has been reached.

The symptoms themselves are the main factors helping one to a successful diagnostic conclusion. Fullness, weight and distension in the epigastrium one-half to three-quarters of an hour after food, relieved by belching or vomiting, are suggestive. The presence of flatulence not relieved by diet in a suspected case is a very strong indication of gall-bladder disease. All the symptoms of uneasiness which force one to relax one's belt or one's clothes, brought on by certain foods, especially those of a greasy nature, with acid regurgitation, heart-burn, gooseflesh, are invariably present in gall-bladder trouble. In rare cases hæmaturia may be amongst the first signs noted, as instanced by the following case.

S.J. was admitted to a public hospital on August 10, 1929, with a history of pain, radiating beneath the right rib margin to the shoulder, of five months' duration. His appetite was good, and there was no history of vomiting, no flatulence, no jaundice, and no clay-coloured stools. Before his admission to hospital he was seen by me in consultation with Dr. Clifton Walker and the late Dr. Leslie Utz; the latter found blood and blood corpuscles in the patient's urine.

On examination, the patient was a strong, healthy looking man, with a tender palpable tumour at the site of the gall-bladder. X-ray examination on August 10 suggested obstruction to the duodenum, which was pulled up under the liver and fixed. (At this time Graham's test was little used.) At operation on August 25 a thickened gall-bladder full of stones was removed; the duodenum was adherent to the gall-bladder.

Once acute inflammation sets in Murphy's sign can be elicited. Vomiting in these cases usually brings on severe prostration. When once an attack of colic occurs, it may be assumed that trouble has been brewing for years and, if the colic is due to the presence of calculi, that a condition exists beyond all medical aid.

It has to be remembered that appendicitis is a common accompaniment of cholecystitis, that ulcer of the stomach and duodenum are often an added complication, and that pancreatitis is frequently present, so that the picture may be greatly obscured and considerable difficulty may be experienced in coming to a correct diagnosis of the primary seat of trouble.

Investigation by several experimenters of the stomach contents alone as a means to diagnosis in these cases has not proved helpful, as hyperacidity, normal acidity and hypoacidity may be met.

The conditions most difficult to differentiate from gall-bladder disease are appendicitis, gastric and duodenal perforation, renal colic, acute pancreatitis, intestinal obstruction (acute ileus), and last, but by no means the least difficult, central pneumonia and diaphragmatic pleurisy on the right side. Both of these last-mentioned conditions often accompany acute gall-bladder infections and are probably due to the free lymphatic drainage of the gall-bladder and to the intimate association of the subdiaphragmatic and superdiaphragmatic lymphatics. Anatomists do not acknowledge this anastomosis, but clinically it does exist. In support of the contention, Inglis some years ago demonstrated at Sydney Hospital a case of malignant disease of the gall-bladder with myriads of secondary deposits on both sides of the diaphragm continuous with each other and passing from the peritoneal to the thoracic cavity and into the right lung.

Complications.

The surgical complications of gall-stones are many and can be alarming. Perforation will always need immediate operation and raises the problem of what should be the *modus operandi* in the presence of acute inflammation of the gall-bladder. "*Festina lente*" is, on the whole, the favoured procedure.

In the case of common duct stones associated with jaundice, the reappearance of coloration in the fæces is a helpful prognostic sign. Stones may be present in the common duct in the absence of jaundice, and Lahey[10] states that in up to 20% of cases of cholelithiasis stones in the common duct are present as well; most surgeons in Australia would consider this figure rather high.

Pancreatitis in a small degree is probably always present in acute cholecystitis. Morley[10] states that in 60% of cases of acute pancreatitis, gall-stones or gall-bladder disease is present.

Fistulæ of the gall-bladder, between the gall-bladder and the duodenum or colon, are usually diagnosed at operation.

Cancer of the gall-bladder has been artificially produced by Leitch,[11] by introducing pebbles into the gall-bladder of guinea-pigs. It has been stated by more than one observer that about five in every hundred persons suffering from gall-stones have cancer of the gall-bladder.

Intestinal obstruction due to gall-stones is a rare event and is seen in cases of large stones which have passed into the small gut.

Treatment.

Treatment should aim at preventing gall-bladder infection by attending to the predisposing factors, such as constipation and bacillary infections of the urinary tract, and by the prescribing of a suitable diet, (*a*) to avoid as far as possible any digestive troubles, (*b*) to keep the cholesterol content of the blood normal, and (*c*) to prevent infection of the biliary tract by intestinal intoxication.

When symptoms of cholecystitis have arrived, dieting may ward off future attacks; but by the time that stones have developed medical treatment can achieve nothing.

and no more disastrous termination confronts the surgeon than cancerous formation on top of cholecystitis when previously a plea had been made for operation.

Operations on the gall-bladder are fraught with great difficulty, and the bad results attributed particularly to cholecystectomy can be traced to faulty technique. The distress and pain suffered in biliary colic, which can be relieved only by morphine at the height of the attack, are considerable, and one attack will make a patient weigh seriously the advantages of an operation rather than face another experience of colic. Attacks tend to follow each other closely and to come on unexpectedly, each one doing considerable damage locally and to the body as a whole, and though the risks of operative procedures are not inconsiderable, operation at least offers more mercy than medicinal measures.

When a gall-bladder has become the seat of disease Nature has little at her command to rid the organ successfully of its infection. As to operations on the gall-bladder in general, one can state definitely that drainage of the gall-bladder is becoming increasingly less frequent and removal more frequent. What operative measure will be adopted will necessarily depend on the type of case that confronts one.

Operation in the Presence of Jaundice.

When the patient is jaundiced, the less done the better. It is interesting to note that, as far back as Sherren's day,[22] in cases of common duct stone without jaundice the mortality rate was 2·5%, and when jaundice was present the rate was 27%.

Before operation saline infusions and calcium chloride and even blood transfusion should be administered. At operation local anæsthesia should be used, means should be adopted to keep up the bodily heat, particularly in the liver area, by warm packs, and drainage of the gall-bladder or common duct should be instituted. After operation fluids must be given freely and the bodily heat maintained, and if necessary blood transfusion again used.

Inhalation anæsthesia which enters into chemical compound with the blood must be avoided. Jaundiced patients tolerate badly ether anæsthesia in particular.

If malignant disease is obstructing the common duct and cholecyst-enterostomy is contemplated, it may be advisable to carry out preliminary drainage. As a rule these patients come to operation too late and the mortality rate is high.

Operation in the Absence of Jaundice.

For non-jaundiced patients, in the absence of complications, cholecystectomy without drainage is the operation of choice.

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ACUTE DISSEMINATED LUPUS ERYTHEMATOSUS, WITH REPORT OF A FATAL CASE.

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PERUSAL of the medical literature reveals that no case of acute disseminated *lupus erythematosus* has been reported in any medical journal in Australia during the past ten years. While a considerable amount of research has been done on this disease in recent years and its presence as a clinical entity has become more widely recognized, we are still in the dark as to its ætiology, and moreover, as to the reason for its high mortality. Unfortunately the case to be described does nothing to throw any extra light on the disease, particularly as no autopsy was performed owing to unavoidable circumstances; but the features of the case are in many respects typical of the salient manifestations of the disease as described by G. Baehr[1] and by Cluxton and Krause.[2] An additional interest, in view of recent reports by Jakobowicz and Bryce[3] and by Krieger *et alii*,[4] lies in the fact that the treatment was affected by the patient's having Rh-negative blood, a complication undiscovered until after two blood transfusions had been given.

The patient was admitted to a Royal Australian Air Force hospital for male and female personnel, where the vast majority of inmates were suffering from comparatively minor complaints, and it was some time before the true nature of the disease process was recognized. For this reason it is necessary to report at some length the progress in the early stages of the illness—that period during which more common diagnoses suggested themselves, only to be revoked in the light of subsequent events.

G. Bahr[1] describes disseminated *lupus erythematosus* as a disease of unknown ætiology, with a striking predilection for young females, and manifested by a prolonged course with fever, the presence of an erythematous rash on the face and parts of the body at some time during the course of the disease, characteristic changes in the vascular system, and usually with a fatal termination. Most observers state that 90% of subacute cases end fatally within five years and 90% of acute cases in twelve months. The generalized form of this disease was first described by Kaposi[5] in 1872.

It is considered by some observers that the underlying cause of the condition may be a tuberculous infection. This was first suggested by Cazenave[6] in 1852, and it was not until 1919 that Barber[7] expressed the opinion that the disease might in some cases be associated with and caused by a streptococcal focus of infection. Roxburgh[8] has described fatal cases in which streptococci were found in the dermis. However, isolation of streptococci from the cutaneous lesions or the manifestation of their presence as a generalized septicæmia is unusual. There is considerable cogency in Gray's[9] observation that the constant uniformity of the microscopic appearances in the disease indicates that it may ultimately be shown that *lupus erythematosus* is caused by a single specific agent as yet unknown.

Cluxton and Krause[10] state that at the present time there are essentially three schools of thought regarding its ætiology. One group, predominantly German, believe that the disease is associated with tuberculosis in most instances. A second group, mostly English, hold that it is due to a septicæmia, probably of streptococcal type. The third group, notably the Americans, favour the varied ætiology (mostly toxic).

All observers are agreed on the characteristic features of the eruption. This is described by E. Rose and D. M. Pillsbury[11] as being polymorphic and usually active. The entire gamut of lesions seen in the erythemata may be encountered; the relationship to *erythema multiforme* has been suggested. The exposed areas of the body are the sites of predilection, but no cutanous or mucous surface is immune. The typical intial lesion appears (usually on the face or nose) as a small, often impalpable, erythematous patch, which usually spreads rapidly. The so-called bat-wing or butterfly configuration across the nose and cheeks is characteristic. The affected area may rapidly extend to include the entire face, ears and neck, and the upper part of the trunk. A wide variety of lesions may appear, simultaneously or in succession—livid, œdematous, urticaria-like or erysipeloid areas, purpura, showers of petechiæ, or reddish-brown to purple macules, èczematoid patches, bullæ, crusted areas or ulcers. The vermilion border of the lips may be affected by œdema, crusting, fissuring or ulceration. While no single skin lesion is characteristic of all cases of acute *lupus erythematosus*, the most common and persistent change is a deeply erythematous swollen patch or plaque of varying size. When this appears on the cheeks, the appearance may strongly suggest erysipelas; but the erythema is usually more livid and progression of the margin more rapid than in the latter condition. When remission occurs, considerable atrophy and scarring or pigmentation may follow the subsidence of the cutaneous lesions.

The pathological changes in the body in this condition are widespread and apparently heterogeneous; but it has been shown that the constant site of the damage, whatever may be the cause, lies in the connective tissues of the body. Thus pericarditis, pleuritis, perisplenitis and perihepatitis are common. About 30% of cases are characterized by a type of endocarditis first described by Libman and Sacks,[12] and consisting of small vegetations not limited to the closure line of the valves, but extending on both sides of the leaflets and down the *chordæ tendineæ*. This is the result of necrosis of the subendothelial collagen tissue. The kidney changes are not unlike those of malignant nephrosclerosis, and, as described by Baehr, Klemperer and Schrifin,[13] the "wire loop" appearance is due to a peculiar hyaline thickening of the walls of the glomerular capillaries, indicating a fibroid degeneration and collagenization of the basement membrane. Similar damage to vascular connective tissue is found in other organs, particularly the spleen. These writers have summed up the vascular lesions as consisting of (i) capillary dilatation with extravasation of blood serum, (ii) proliferative endothelial vascular lesions with thrombus formation, and (iii) degenerative or necrotizing lesions in the walls of the capillaries, arterioles and venules, often with hæmorrhage into adjacent tissues. Examination of the involved skin reveals degenerative changes in the collagenous tissue of the upper corium culminating in fibrinoid degeneration. The smaller blood vessels may also participate, with resultant epithelial damage and hæmorrhages therein. The lymph nodes are enlarged in about one-half of the cases.

The salient clinical manifestations are (i) prolonged irregular fever, (ii) depression of bone marrow function, resulting in leucopenia, moderate hypochromic anæmia and thrombocytopenia, (iii) clinical evidence of vascular alterations in the skin, the kidneys and other viscera, and (iv) a tendency to recurrent involvement of synovial and serous membranes (polyarthritis, pleuritis, pericarditis).

Report of a Fatal Case.

Corporal K.J.S., a member of the Women's Australian Auxiliary Air Force, aged twenty-eight years, single, was admitted to a Royal Australian Air Force hospital on July 6, 1944. She was complaining of a cough with yellow sputum which had been present for a week. For one day she had had sweats and felt feverish, but she had no pain in the chest nor sore throat. She had noticed an enlarged gland in the left axilla two days before her admission to hospital. For some two months she had had a saddle-shaped erythematous rash on her nose and cheeks, but this had waxed and waned with treatment.

On examination, her temperature was 102° F. and her pulse rate 100 per minute, and her respirations numbered 22 per minute. Physical examination of the chest revealed no abnormality, the throat was normal, no mass or viscus was palpable in the abdomen, and a few slightly enlarged glands were present in the left axilla. The rash on her face was saddle-shaped and covered the bridge of the nose and spread symmetrically onto both cheeks. It was a dull red colour, was slightly raised and consisted of confluent macules. It also spread to the ear lobes. No other areas were affected.

A provisional diagnosis was made of influenza or early glandular fever, and simple treatment was commenced, consisting of the administration of an expectorant mixture, a linctus and "A.P.C." (ten grains every four hours). The same night her temperature rose to 104° F. and she felt very miserable. On examination, it was found that the *alæ nasæ* were working and that the cough was more severe; she was bringing up thick yellow sputum. The next day her temperature was still high (104° F.) and she was very ill. On examination of the chest, some diminished breath sounds and diminished vocal resonance were noted at the base of the left lung, with crepitations on deep breathing. She was treated with sulphathiazole tablets, two grammes being given as an initial dose and one gramme every four hours thereafter. The leucocytes numbered 7,500 per cubic millimetre of blood (see Table I).

TABLE I.

Date.	Erythrocytes per Cubic Millimetre.	Hæmoglobin Value *per Centum.*	Colour Index.	Leucocytes per Cubic Millimetre.	Neutrophile Cells *per Centum.*	Eosinophile Cells *per Centum.*	Monocytes *per Centum.*	Lymphocytes *per Centum.*
7.7.44				7,500	70·0	1·0	3·0	26·0
13.7.44	3,810,000	85·0		3,450				
15.7.44				4,450	68·0	0·9	3·2	27·3
18.7.44				5,100				
26.7.44	3,530,000	85·0	1·1	8,650	83·0	0·5	2·0	14·0
2.8.44	2,970,000	57·0		3,980	70·0	0·5	2·0	27·5
8.8.44	4,380,000	79·0	0·9	8,650	84·0		2·5	13·5
15.8.44	4,720,000	85·0						
22.8.44	3,900,000	75·0	0·81					
28.8.44	3,010,000	68·0	1·0	4,050	72·0	1·0	3·0	24·0
2.9.44	4,820,000	90·0		7,800	75·0	1·0	3·0	21·0
10.9.44				5,750	55·0	1·0	5·0	38·5
16.9.44	2,390,000	45·0		6,150	70·0	1·0	4·0	25·0
23.9.44	1,970,000	45·0						
10.10.44		26·0						

On the third day the temperature was still elevated and the patient was very ill. Examination of the lungs revealed diminished percussion note and diminished breath sounds at the base of the right lung, with coarse crepitations and increased vocal resonance. A diagnosis of right basal pneumonia was made and sulphathiazole therapy continued. It was also found that a slight redness of the skin was present on the back of the chest and on the back of the arms, in the form of slightly raised, disk-like lesions of a purplish-red colour and not as yet confluent.

On July 9 her condition had not improved, and she appeared somewhat cyanosed. Her systolic blood pressure was 105 millimetres of mercury and her diastolic pressure 80, and she did not appear clinically to be responding to "M & B 760". On July 10 her temperature was still elevated to 104° F. and she was very cyanosed. Examination of the chest revealed vague signs at the base of the left lung also—namely, some diminished breath sounds and coarse crepitations. It was obvious that the condition was not ordinary lobar pneumonia, and also that it was not responding to sulphathiazole. A specimen of sputum was examined pathologically; no pathogenic pneumococci were present. A blood examination revealed that the leucocytes numbered 6,000 per cubic millimetre (see Table I). Oxygen was ordered for ten minutes every hour, and it was decided to commence penicillin therapy and discontinue sulphathiazole treatment, as there had been a fall in leucocyte count and no apparent response. Penicillin was given in doses of 10,000 units intramuscularly every hour for five hours at night and in the morning.

On July 11 the patient looked and felt better; but her temperature was still elevated to 103° F., her pulse rate was 102 per minute, her respirations numbered 24 per minute, and her blood pressure was 98 millimetres of mercury (systolic) and 64 (diastolic). The physical signs in the chest were unchanged.

On July 12 her condition seemed to be improved and her temperature was down to 101° F., but examination of the lungs revealed an increase in the adventitious signs at both bases, many moist râles being heard. A large quantity of sputum was still being coughed up. On July 13 her temperature was down to 100° F.; but the patient was drowsy and apathetic, and was still coughing up large quantities of sputum. Blood examination was repeated and revealed a considerable fall in the number of leucocytes to 3,450 per cubic millimetre (Table I). Clinical examination of the lungs revealed no change.

On July 14 there was no change for the better in the patient's condition, and penicillin treatment was discontinued after 500,000 units had been given. On July 15 her condition was unchanged, and the following investigations were set in train: (i) cultural examination of the blood, (ii) a Widal test, (iii) microscopic examination of the urine, (iv) X-ray examination of the chest with a portable machine, (v) a blood count. The X-ray examination revealed no evidence of consolidation. The urine examination revealed numerous pus cells, and *Staphylococcus albus* was grown in culture. The leucocytes numbered 4,450 per cubic millimetre (Table I). On July 17 the patient felt much better, and her temperature reached normal for the first time. Examination of lungs revealed widspread râles at both bases posteriorly.

On July 18 the rash previously described had increased in intensity, and was visible on both arms, on the back and on the buttocks. It was an erythematous papulo-macular rash, and the bat's wing area on the face was particularly pronounced. Examination of the chest revealed fine medium râles over the lower lobes of both lungs posteriorly. On July 19 the temperature was normal for the twenty-four hour period for the first time, and the patient felt better.

On July 22 the patient's temperature had risen again to 102° F. and she still had severe cough and sputum. The physical signs in the chest were unchanged. The result of the agglutination tests was negative for *Bacterium typhosum* H and O and for *Bacterium paratyphosum* A and B, and also for *Brucella abortus*. On July 24, close questioning of the patient revealed some relevant past history. She had had frequent colds with cough and sputum prior to a bilateral radical antrostomy ten years earlier. She had been comparatively free for six months, and had then suffered an annual cold with cough and copious sputum for about two months each year. She found that posture altered her desire to cough, and that she brought up more sputum in a position with her head lower than her feet.

The physical signs were unchanged, and it was considered that there might have been a bronchiectatic basis for her condition, with secondary infection. Sputum examination again gave negative results for pathogenic organisms as well as for fuso-spirochætes. A further X-ray film of the chest revealed slightly increased markings at the base of the left lung, but no evidence of consolidation. The attempt at culture from the blood on July 15 yielded no growth of organisms.

On July 25 the patient's temperature was again about 102° F., and it was decided to recommence penicillin treatment. This was done on July 26, at the rate of 100,000 units per day as before. No improvement was noted over the next few days, and the frequent injections were causing severe distress. The rash was spreading and appeared very angry; it had appeared on the elbows and knees and was commencing on the fingers of both hands. It was somewhat painful to pressure, and required protective dressings. Microscopic examination of the urine revealed no pus, and attempted culture yielded no growth of organisms.

On August 1 blood examination revealed considerable anæmia; the erythrocytes numbered 2,970,000 per cubic millimetre, the hæmoglobin value was 57% on the Haldane scale, and the leucocytes numbered 3,980 per cubic millimetre. Penicillin treatment was discontinued after 650,000 units had been given and a transfusion of three pints of blood by the slow-drip method was commenced on August 6. Some difficulty was encountered with clotting in the transfusion apparatus, and only two pints of blood were given. However, on August 7 the patient felt much better, and examination revealed slight diminution of moist sounds in the chest.

Over the course of the next week the patient felt better, but still continued to cough and bring up profuse sputum. A Mantoux test was performed; the patient reacted to old tuberculin diluted 1/100, but not when it was diluted 1/1,000. Postural drainage was tried, but was unsuccessful. During this period the transfusion wound in her leg became infected and was subsequently slow to heal. The rash was slightly less pronounced, but it had appeared on her finger tips, causing some pain on pressure. The skin of these areas peeled off, and her nails became brittle and deformed. Examination of the sputum again failed to reveal tubercle bacilli or actinomyces. A blood count revealed that the number of erythrocytes had improved to 4,380,000 per cubic millimetre, the hæmoglobin value to 79% and the number of leucocytes to 8,650 per cubic millimetre. Dark-ground examination of the sputum on August 14 revealed an occasional spirochæte of Vincent's type.

During this period the patient's temperature was of the "swinging" type. On August 16 a further X-ray examination was made and the following report was obtained: "Increased bronchovascular markings at both bases suggesting inflammatory change, possibly on a bronchiectatic basis." On August 21 her temperature was again mounting to 102° F. and she was complaining of pain in the joints of the upper limbs. These pains were present in the left shoulder, in the elbows, and in the metacarpo-phalangeal and interphalangeal joints of both hands. No objective joint changes could be found on examination, nor any limitation of movement.

At this stage, despite the continued fever and joint pains, the patient felt slightly better, her cough was less severe and she was producing less sputum. Examination of the chest revealed that the râles were now limited to the extreme bases of both lungs. In view of the decrease in the chest signs and in the absence of any other definite focus of infection it was considered that the condition was probably disseminated *lupus erythematosus*. Treatment with *Liquor Arsenicalis* was commenced on August 23.

A further blood examination on August 23 revealed that the erythrocytes numbered 3,900,000 per cubic millimetre and that the hæmoglobin value was 75% on the Haldane scale. X-ray examination of the hands revealed no abnormality. On August 27 the erythema was more pronounced and more widespread on the face, arms, legs, back and buttocks, and the hands were again becoming affected. The joint pains had lessened to a considerable extent. The *Liquor Arsenicalis* treatment was discontinued, and quinine therapy (five grains three times a day) was instituted. Examination revealed some small palpable glands in both axillæ, and auscultation of the chest revealed a few scattered râles at the bases of both lungs. A blood count on August 28 revealed that the erythrocytes numbered 3,010,000 and the leucocytes 4,050 per cubic millimetre, the hæmoglobin value being 68%. The erythrocytes were at the microcytic mean.

Blood transfusion into the arm was commenced on September 1. Three pints of blood were given, rather more rapidly than by "slow drip", in order to overcome the difficulty previously encountered with clotting. After the transfusion a blood examination revealed that the erythrocytes numbered 4,820,000 and the leucocytes 7,800 per cubic millimetre, and the hæmoglobin value was 90%. On September 2 treatment with "M & B 125" was begun, one

tablet being given every four hours. On September 4 the patient was slightly jaundiced and nauseated. Microscopic examination of the dark reddish-brown urine revealed fairly numerous pus cells, much albumin and some acetone and diacetic acid present; no bile salts, no sugar and no pigments were seen. On September 5 examination of the urine revealed the same findings, and a few erythrocytes as well. Over the course of the next few days the urine continued to contain a large amount of albumin, but the patient felt reasonably well; "M & B 125" treatment was discontinued. On September 9 the rash was less pronounced in all areas and the albumin had disappeared from the urine. It was decided to commence gold therapy with sodium thiosulphate, and also to give injections of "Anahæmin". This treatment was commenced on September 11; a dose of 0·005 milligramme of "Myocrisin" was given intramuscularly and 0·05 gramme of sodium thiosulphate intravenously. Two cubic centimetres of "Anahæmin" were given intramuscularly.

Urine examinations were made over the next few days, the results being as follows. On September 11 the urine contained numerous pus cells and a few non-mobile bacilli, but no albumin. On September 13 the urine contained numerous pus cells, an occasional erythrocyte and some albumin. On September 15 the urine contained numerous pus cells, an occasional cellular cast and much albumin.

On September 16 the patient was feeling comparatively well; but blood examination revealed a considerable deterioration; the erythrocytes numbered 2,390,000 and the leucocytes 6,150 per cubic millimetre and the hæmoglobin value was 45%. On September 20 a blood transfusion was commenced by "slow drip" into the arm. It was intended to give three pints, but after 800 cubic centimetres had been run in over a period of ten hours it became obvious that some incompatibility existed *in vivo*, as the patient's general condition suddenly deteriorated. She became jaundiced, her temperature rose to 105·8° F. and her pulse rate was 126 per minute. She was dyspnœic and vomiting occasionally. The next day, September 21, her condition was poor, although the temperature was lower. Considerable jaundice and cyanosis were present, and her urine, which was being passed in fair quantities, was bright red. This was found to be due to hæmoglobinuria, as only a few erythrocytes were found on microscopic examination. On September 22 her general condition was still very bad, frequent vomiting, cyanosis and the passage of red urine being the salient features. Her liver was palpable one finger's breadth below the costal margin, but the spleen was not felt. All treatment was discontinued except administration of a simple potassium citrate mixture. On September 23 blood examination revealed that the erythrocytes numbered 1,970,000 per cubic millimetre and the hæmoglobin value was 45%, but her general condition was much improved. In the night she had an epileptiform convulsive seizure followed by a period of unconsciousness, but examination on September 24 revealed no abnormal neurological signs.

A steady downward course was commenced from this time onwards. There was no great increase in the rash, but her scalp was quite bald in patches. Over the next five days she had three further convulsive seizures and her urinary output decreased to as little as four ounces on some days, albumin being present. On October 3 the patient's general condition had deteriorated and her temperature was subnormal. During the night of October 3 she had a severe epistaxis, which was controlled by plugging the nostrils.

It appeared that the patient would die at any time over the course of the next few days; but her faculties were retained until October 9, when she became lethargic and was voiding urine and fæces into the bed. On October 10 her hæmoglobin value was 26%, and it was decided to try a further blood transfusion. In view of the result of the previous transfusion, it was suspected that the patient's blood was Rh-negative and that Rh antibodies had developed in her serum as a result of the first transfusion. Such was found to be the case, and on October 12 transfusion by the slow-drip method was commenced with Rh-negative blood of group O (IV). After four and a half pints of Rh-negative blood had been given, pulmonary œdema and an irregular pulse developed. Her condition was not regarded as due to any incompatibility of blood or to the rate of administration or the quantity administered, but to myocardial degeneration resulting from the long duration of the disease, with its associated toxæmia. Death occurred at 3.45 p.m. on October 14.

Discussion.

In the case described irregular fever went on for a period of 102 days. There were only two occasions in the first twelve weeks on which the temperature was normal for a period of twenty-four hours. It was amazing to the observers that the patient's general condition could have kept up for so long under these conditions. The early fever with cough and vague signs in the chest suggested a diagnosis of pneumonia, as quite a number of personnel were suffering from lobar pneumonia at the time. However, there was a failure to respond to sulphathiazole after four days, and it was decided to commence penicillin therapy. The steady fall in temperature described gives a false impression as regards the response to penicillin. To an observer in close association with the case the clinical response was not favourable, so penicillin therapy was stopped. It appeared then as though the condition might be a primary atypical pneumonia, which is known to be unresponsive to chemotherapy. This was ruled out when an X-ray film of the chest was taken with a portable machine, as the picture bore no resemblance to the fleecy opacities seen in this condition. It was necessary to exclude all other causes of pyrexia of unknown origin, and investigations excluded typhoid fever, paratyphoid fever A and B, septicæmia and undulant fever. At this stage there was some evidence of urinary tract infection as revealed by microscopic examination of the urine.

The subsequent revelation of past antral infection led to the feeling that the underlying pathology of the condition might have been bronchiectatic, with secondary infection of the adjacent pulmonary tissue, and the fact that this possibility was not ruled out by subsequent autopsy leaves some doubt in my mind that such a condition was not present. However, pulmonary complications such as bronchopneumonia, lobar pneumonia, abscess, gangrene and atelectasis are common in disseminated *lupus erythematosus*. Failure of the patient to respond to further penicillin therapy, an exacerbation and spread of the rash, the persistence of the pulmonic condition, and the oncoming of normocytic anæmia with leucopenia led to the tentative diagnosis of disseminated *lupus erythematosus*. The subsequent clinical picture as described amply bore out this diagnosis.

Reference to Table I shows the progressive anæmia which developed despite blood transfusions, and the leucocyte count always revealed leucopenia, relative or absolute. This was evidence of diminished bone marrow function, although the anæmia was doubtless increased subsequently by hæmolysis due to the fact that the patient's blood was Rh-negative—a fact which was unsuspected at the time. What took place at the second and third blood transfusions is clear in retrospect. Although the patient had blood of group O (IV), it was noticed on typing her blood with group O (IV) blood from donors that after an hour in some cases slight agglutination was present. These donors were not used; but the blood that she was given was probably Rh-positive and this resulted in the formation of Rh antibodies in the patient's blood. Strangely enough, typing for the second transfusion did not reveal even delayed agglutination, and the hæmolysis, manifested by jaundice and hæmoglobinuria, was regarded at the time as being due to the fact that the transfusion was given rather too rapidly. This transfusion resulted in the formation of a higher titre of Rh antibodies in the patient's blood, and there were early and serious results from hæmolysis after 800 cubic centimetres of group O (IV) blood had been transfused slowly on the third occasion. The end result of these transfusions was worse than before; the anæmia was quite profound and the kidneys had great trouble to excrete the broken-down products. This was doubtless accentuated by the damage that was being done to the kidneys by the inexorable disease process, and it appeared that further transfusions would be fatal. However, the transfusion just prior to death was carried out with group O (IV) Rh-negative blood, and no reaction attributable to this transfusion occurred. Examination of the patient's blood prior to this transfusion was carried out by Dr. Vera Krieger, who reported that Rh antibodies were present in the serum in a very high titre.

Throughout the course of the illness the severity of the rash rather characteristically waxed and waned. The dark purple mottled appearance of the rash on the face at various periods certainly suggests that hæmorrhages were taking place into the skin. Only for a brief period were there joint manifestations, and when present they were entirely subjective, not giving rise to any naked-eye or

radiological changes. This is rather in contradistinction to the cases described by American writers, in which arthralgia, often acute, has been an early and pronounced feature of the disease. Adequate explanation can be given for the convulsions suffered by the patient towards the close of the disease, on the basis of cerebral involvement. This may be a chronic meningoencephalitis as described by Kiel,[13] or an actual encephalomalacia due to plugging of cerebral vessels as described by Jarcho.[14] At no stage was there clinical evidence of an exudate into any of the serous cavities.

Treatment.

The high mortality of acute disseminated *lupus erythematosus* is ample evidence of the inefficiency of treatment. However, many varied therapeutic agents have been described in the literature.

A. L. Weiner[15] describes four cases of disseminated *lupus erythematosus* treated with sulphanilamide. In two cases the disease was arrested, one patient being followed up for nearly two years and the other only for several months. In the other two cases the drug was given only in the terminal stages and no effect was noted, death ensuing. Weiner states that in the sulphanilamide therapy of *lupus erythematosus* the drug should be administered early in the disease and in large doses, and he does not consider that the leucopenia associated with the disease is a contra-indication to the use of this drug. Its use is combined with the administration of frequent small blood transfusions. Other writers have reported no success with sulphanilamide therapy.[16]

There is no doubt that exposure to sunlight and ultra-violet rays has a deleterious effect. It was noticed in the case described that when the patient exposed her face and chest to the sun, even through the window, her rash flared up and her temperature rose. Exposure to X rays is also condemned;[17] but it is stated that Röntgen irradiation of the ovaries warrants a trial (Contratto and Levine). Eradication of septic foci is to be avoided during the acute phase of the disease, and even in a period of remission is to be undertaken warily.

Liver extract has been used as a therapeutic agent by Cornbleet.[18] He describes eleven patients with cutaneous lesions, a chronic discoid type of *lupus erythematosus*, who were given injections of liver extract (two cubic centimetres) three times a week. The treatment in each case was carried out for from three weeks to four months. There appeared to be partial reduction in the lesions of three patients followed by recrudescence. In the other cases the lesions either did not diminish at all or extended.

There appears to be some difference of opinion regarding the value of heavy metals in the treatment of this condition. R. M. B. McKenna[19] states that the acute fulminant type of disseminated *lupus erythematosus* may be treated by injections of bismuth in the absence of grave systemic disturbances, and even injections of gold may be used cautiously. This does not appear to be supported by American authorities, who are of the opinion that gold should never be used in the acute form of the disease.

It is difficult to state definitely whether the one small gold injection given to the patient under discussion had any harmful effect. No benefit would be expected from one injection, and as a severe transfusion reaction occurred at the time when the second gold injection was due, this form of therapy was abandoned. Had it been possible to keep up the patient's general condition with transfusions and the leucocyte count with "Pentnucleotide", it is thought that this form of therapy might have been of some value.

The steady downward progress of the patient described amply bears out the general opinion held by most observers of the high mortality of acute disseminated *lupus erythematosus*. It is possible that in this condition, as in many others, it is exceedingly difficult to treat a disease process of which the ætiology is not definitely determined.

Summary.

1. Acute disseminated *lupus erythematosus* is described.
2. A brief survey of the current literature is given.
3. A fatal case is reported, the patient being a member of the Women's Auxiliary Australian Air Force.
4. The differential diagnosis arising during the illness is discussed.
5. Some modes of treatment are discussed.

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Reports of Cases.

A CASE OF IDIOPATHIC ACUTE DISSEMINATED ENCEPHALOMYELITIS ("ACUTE PERIVASCULAR MYELINOCLASIS").

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IN recent years considerable interest has been shown in the condition known as "acute disseminated encephalomyelitis". As this title has been applied to several diseases closely resembling each other clinically, but readily distinguishable pathologically, Turnbull[1] prefers the term

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"disseminated encephalomyelitis of the post-vaccinal type". On the other hand Marsden and Hurst[2] have adopted the appellation of "acute perivascular myelinoclasis". The disease has been reported in association with smallpox,[3][4] measles[4][5][6] and respiratory infection,[7][8][9] and subsequent to vaccination,[10][11] the administration of sulphanilamide[12] and antirabic treatment.[13][14][15] In addition a number of so-called "spontaneous" cases have been referred to,[1][4][16] in which there was no apparent precipitating factor.

The clinical and pathological features of the idiopathic type of acute disseminated encephalomyelitis have been summarized by Kinnier Wilson[16] as follows:

> Perhaps occurring mostly in children or juveniles, the "disease" begins abruptly with headache, fever or other infective signs, to which focal cerebral, cerebellar, pontobulbar, spinal, radicular or peripheral nerve symptoms are added, in any combination, or singly. Pains and paræsthesiæ in limbs, trunk or elsewhere, with objective diminution or loss of sensory conduction, are not uncommon, and sphincteric trouble may supervene. Fits, delirium and coma have been observed, as well as optic neuritis and severe meningitic reactions. . . .
>
> Set over against this clinical diversity, however, is a claim for relative pathological unity. Scattered richly through brain and cord, chiefly in white substance, are larger or smaller foci (at times confluent) of acute myelin denudation, with or without axonal neurolysis; at their centres or in their vicinity lie engorged vessels showing a variable degree of hyperplastic reaction; here, too, overgrowth, fibrillar and cellular, on the part of both macro- and microglia can be seen. Similar mesodermal change often affects the meninges. Neural parenchyma otherwise is little damaged, except where patches develop in the more concentrated regions (brain-stem, cord).

Although a considerable number of cases of the disease has been recorded, in only a small proportion have pathological examinations been carried out. Of these cases, histologically, extremely few resemble closely my case; they include the case of Krabbe,[17] the case referred to briefly by Wohlwill,[4] and perhaps the second cases of Greenfield[7] and of Davison and Brock.[9] In view of its rarity, therefore, the clinical and histopathological findings in the present case are reported.

Clinical Record.

R.D.W., a schoolboy, aged seven years, was admitted to the Adelaide Children's Hospital on September 7, 1943, under the care of Dr. E. Britten Jones. For a month he had been drowsy. On occasions during this period he had been very energetic; for instance, when out walking he would race ahead of the other members of the party; afterwards, however, he would appear to be unduly tired. For a fortnight before his admission to hospital he had complained of pains in his abdomen just below the costal margins. He appeared to feel the cold and was rather irritable, but was still able to go to school. His appetite gradually failed. Two days prior to his admission to hospital he became extremely sleepy and would not speak. He was put to bed. On the morning of his admission the child was semi-comatose; he still complained of pains in his abdomen. His private medical attendant elicited Kernig's sign.

On examination of the patient, his temperature was 98·6° F., his pulse rate was 124 per minute and his respirations numbered 26 per minute. He was comatose. The pupils were equal and reacted to light; there was no evidence of papilloedema or of chorioidal tubercles. The tonsils were enlarged and the pharynx was reddened. An occasional rhonchus was heard over the posterior aspect of both lung fields. The plantar reflexes were extensor in type. The blood contained 100 milligrammes of glucose per 100 cubic centimetres. Lumbar puncture yielded clear cerebro-spinal fluid under a pressure of 150 millimetres of water; of the 28 cells per cubic millimetre present all were lymphocytes; the fluid contained 720 milligrammes of chlorides and 25 milligrammes of protein per 100 cubic centimetres; sugar was present, the amount of globulin was not increased, no tubercle bacilli were found on examination of a direct smear, and there was no coagulum when the fluid was allowed to stand. The fluid was sterile. Examination of the urine revealed severe acetonuria but no glycosuria. During the day the boy had several "spasms" of the arms and legs. Occasionally he sweated.

On September 8 the child's limbs were rigid and his pupils would not react to light. The urine contained no cells, but *Staphylococcus albus* was grown on culture. The intradermal tuberculin test produced no reaction with a dilution of one in ten. The patient's blood contained 4,800,000 erythrocytes and 10,800 white blood cells per cubic millimetre; of the latter 79% were polymorphonuclear leucocytes, 16% lymphocytes and 5% monocytes; platelets were plentiful; the red blood corpuscles showed some pallor; the hæmoglobin value was 81%; the colour index was 0·7. The blood picture was considered to be suggestive of iron deficiency.

On the following day the boy's temperature was elevated to 103·2° F. There were occasional attacks of coughing with the expectoration of mucous sputum.

On September 10 the patient's breathing was stertorous and he still had spasms of coughing. His plantar reflexes were extensor in type. The fundi of both eyes were normal. The cerebro-spinal fluid obtained on lumbar puncture contained 32 white cells per cubic millimetre, of which 98% were lymphocytes and the remainder polymorphonuclear leucocytes; the fluid contained 760 milligrammes of chlorides and 25 milligrammes of protein per 100 cubic centimetres; the globulin content was not increased, sugar was present and the fluid was sterile. Towards evening on September 11 the child's general condition deteriorated. Signs and symptoms of hypostatic pneumonia were clearly evident. Death occurred at 9.15 a.m. on September 12, 1943.

Additional information obtained from the patient's parents after his death revealed that there were seven other children in the family, all of whom had remained healthy. The child had contracted mumps about six months before his admission to hospital, but had made a complete recovery. He had never suffered from asthma, hay fever or urticaria.

Post-Mortem Examination.

Autopsy was performed by Dr. E. B. Sims on September 13, 1943, about twenty-four hours after death. Except for the cerebral condition, the only abnormalities disclosed were hypostatic pneumonia and the fact that the spleen was soft and somewhat enlarged and contained small pale yellow areas.

Attempts to Transmit the Disease to Animals.

One monkey (*Macaca irus*) was inoculated with one cubic centimetre intracerebrally and ten cubic centimetres intramuscularly of a 10% suspension of brain substance. Six mice were inoculated with 0·025 cubic centimetre of the same suspension intracerebrally. The results were negative.

Neuropathological Report.

Macroscopic Examination.

Externally the brain appeared to be normal. Examination of sections of the brain after its fixation *en masse* in 10% formol-saline solution revealed scattered, small, greyish-pink, translucent, gelatinous-looking areas, most evident in the white matter forming each *centrum semiovale*. The smallest lesions were just visible to the naked eye, while the largest were about one millimetre in their transverse diameter; on an average they measured about 0·5 millimetre. With the aid of a hand lens it was seen that the lesions were arranged perivascularly.

Microscopic Examination.

Character of Lesions.

Pia-arachnoid infiltration was mild and extremely patchy. It was limited almost entirely to the base of the brain, and occurred mainly on the ventral aspect of the brain stem, in the cerebello-pontine angle, between the cerebellar folia and in the depths of the hippocampal fissure. The cells of the infiltrate consisted of lymphocytes together with occasional adventitial cells and a few eosinophile polymorphonuclear leucocytes.

Within the central nervous system the most outstanding feature of the pathological process was perivascular (extra-adventitial) demyelination. Associated with it were œdema, extraadventitial necrosis of axis cylinders, and perivascular, extraadventitial and diffuse infiltration.

In myelin sheath preparations (Weil-Weigert), two types of lesion were apparent. The first consisted of clear-cut zones of complete demyelination, perivascular (extra-adventitial) in distribution (Figure I). In severely affected regions the zones measured from three to five times the diameter of the vessels they encircled. Occasionally the foci coalesced to form larger areas (Figure II). Macroscopically the second type of lesion appeared as a diffuse area of pallor which extended centrifugally from the extra-adventitial foci (Figure I). In a few instances the diffuse lesions appeared to occur independently, but this may have been an artefact dependent upon the plane of section. Microscopic examination of the areas of diffuse pallor disclosed

pronounced swelling and varicosity of the myelin sheaths, which stained less intensely than normal. In places the sheaths were separated one from another.

Necrosis of axis cylinders occurred only in zones of total loss of myelin. In intensity it was only moderate; the thicker axones were affected less than the thinner (Figure III).

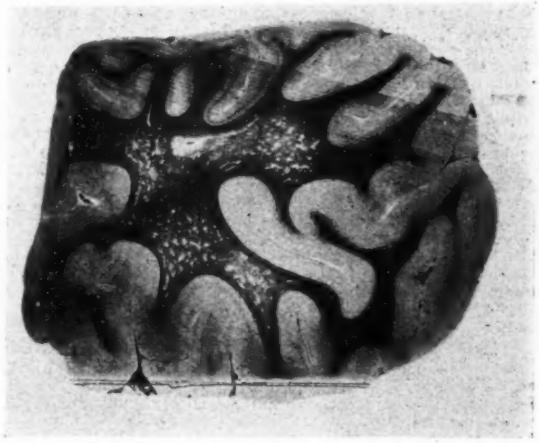

FIGURE I.

Right occipital lobe. Frontal section passing through tip of posterior horn of right lateral ventricle. Perivascular (extra-adventitial) and diffuse demyelination. (There is an artefact at one margin brought about by the junction of two cover slips.) Celloidin section. (Weil-Weigert method for myelin; × 1¼.)

Perivascular infiltration was variable in degree. Usually it was moderate, ranging from two to three cells in thickness. In a few instances "cuffs" up to eight cells in thickness were observed. The infiltrate was composed of lymphocytes, com-

FIGURE II.

Centrum semiovale. Perivascular (extraadventitial) and diffuse demyelination. Celloidin section. (Weil-Weigert method for myelin; × 50.)

pound granular corpuscles, occasional plasma cells and a few eosinophile polymorphonuclear leucocytes. Usually perivascular "cuffing" occurred in association with extra-adventitial infiltration; sometimes, however, it existed independently.

Extraadventitial infiltration was of moderate to severe intensity. In distribution it corresponded to the zones of complete demyelination (Figure IV). The infiltrate was formed mainly by polymorphic microglial cells in various stages of transition to lipoid-containing compound granular corpuscles and by swollen astrocytes (*gemästete* cells). Most of the former were at the "rod-cell" stage; fully formed

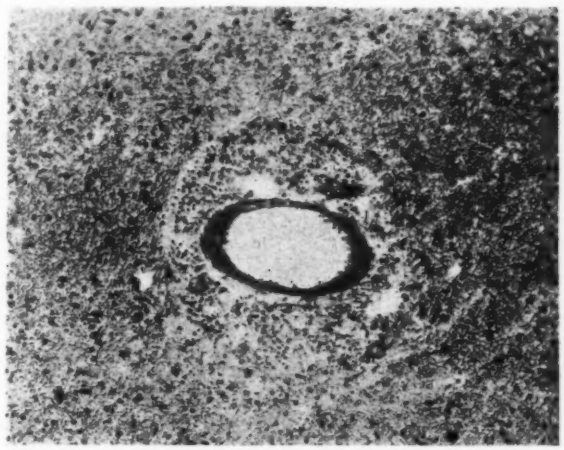

FIGURE III.

Centrum semiovale. Perivascular necrosis of axis cylinders. Frozen section. (Bielschowsky method; × 110.)

gitter cells were less common. In the affected areas the oligodendroglia had the appearances of acute swelling; that is, the nuclei were swollen and were surrounded by a halo. In addition, a few plasma cells were occasionally seen, mainly in regions of transition between diffuse and extra-adventitial infiltration. Occasionally the plasma cells were binuclear.

Corresponding to the presence of *gemästete* cells in the extraadventitial and diffuse infiltrates, in sections stained by Anderson's Victoria blue method, there was a slight increase in glial fibres. This increase was most apparent in the nervous tissues adjacent to vessels (Figure V).

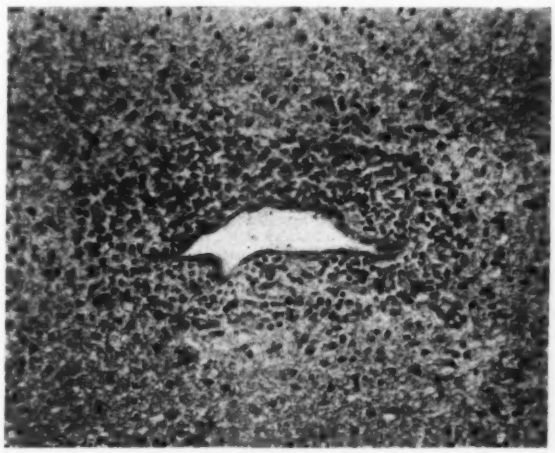

FIGURE IV.

Centrum semiovale. Extraadventitial infiltration. Paraffin section. (Hæmatoxylin and eosin; × 160.)

The vessels surrounded by the lesions were moderately dilatated. The majority were venules or smaller branches. Occasionally arterioles were affected; in such instances the lesion was limited either to perivascular infiltration, or if demyelination occurred the "cuffs" were narrower than those associated with venules, the accompanying extraadventitial

infiltration was less intense, and the adjacent diffuse infiltration was usually absent.

The endothelium lining the vessels was sometimes slightly swollen. True thrombosis of vessels did not occur; in a few instances the red blood corpuscles within the lumina were in a process of being fused together, but these appearances were considered to be the result of changes occurring at the time of death or *post mortem*. At no time was fibrin detected, and there was no attempt at organization.

On a few occasions a moderate increase of the collagen fibres of the adventitial sheath of the vessels was noted. Degenerative changes in the vessel walls were rare. Hæmorrhages were extremely uncommon; in a prolonged search only one was found, and this was minute.

It should be emphasized that longitudinally, the perivascular lesions described above could be traced along the whole extent of the vessel which appeared in any particular section.

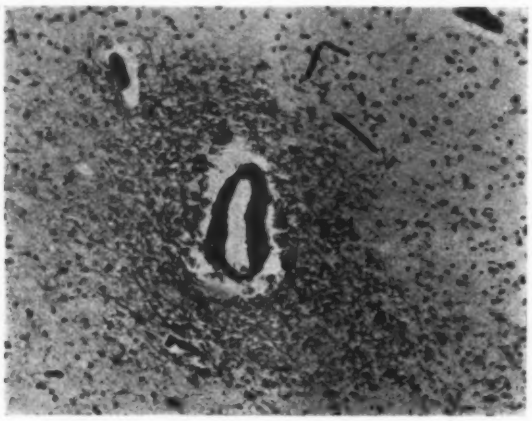

FIGURE V.

Centrum semiovale. Extraadventitial increase in glial fibres. Frozen section. (Anderson's Victoria blue method; × 110.)

Distribution of Lesions.

Cerebrum.—The brunt of the pathological process was borne by the *centrum semiovale*. In the right cerebral hemisphere there was a tendency for the dorsal half to be affected more than the ventral half; in the left cerebral hemisphere the opposite was the case. All lobes—frontal, parietal *et cetera*—were fairly evenly involved. The lesions were extremely severe and affected clumps of vessels ranging in number from about 50 to 100 (Figure I). The collections of vessels together with the extraadventitial and diffuse demyelination related to them formed areas which measured up to two centimetres in their greatest diameter. Most of the lesions were separated from the cortex by intervening normal tissue. A few small vessels with mild perivascular infiltration were seen at the junction of cortex with white matter. Pathological changes in the cortex were mild. Except in one minor instance, demyelinating lesions did not occur. Occasional cortical neurones showed swelling, central chromatolysis, and nuclear and nucleolar eccentricity. These appearances were characteristic of retrograde degeneration, and were attributed to interruption of axones in their course through the *centrum semiovale*. A few cortical vessels showed mild perivascular infiltration, notably in the supramarginal gyrus, the superior temporal gyrus and one of the occipital gyri of the right cerebral hemisphere, and in the *gyrus rectus*, one of the orbital gyri, the anterior central gyrus, the hippocampal gyrus and the inferior temporal gyrus of the left cerebral hemisphere. Almost all of the affected vessels lay in the deeper cortical laminæ. Lesions of moderate intensity was observed in the thalamus. In numerous vessels moderate (sometimes severe) perivascular infiltration was noted, together with a mild to moderate degree of perivascular demyelination and associated changes. On the left side there were a few vessels with moderate perivascular demyelination in the region of the claustrum and adjacent external capsule. One small area of focal infiltration was present also. In the subependymal tissue adjacent to the left lateral ventricle, mainly in the region of the *stria terminalis*, occasional vessels with moderate perivascular infiltration were observed. No lesions were found in the *corpus callosum*, the optic tracts, the *corpus striatum*, the lateral geniculate body, the *cornu Ammonis*, the *gyrus dentatus*, the ependyma and the chorioid plexuses.

Brain Stem and Cerebellum.—In the mid-brain a few vessels with mild perivascular infiltration were seen in the region of the red nucleus. In the *basis pedunculi* of each side there were a few vessels with mild perivascular infiltration; on the right side two of the vessels showed mild perivascular demyelination as well. In the tegmentum there were a moderate number of vessels with moderate perivascular infiltration; a few exhibited slight perivascular demyelination. Lesions were less extensive and intensive in the cerebellum and pons than in the mid-brain. Perivascular demyelination was confined to the white matter enclosed by the left dentate nucleus (here, there was also some diffuse demyelination), to the right restiform body and to scattered vessels in the reticular substance, especially just dorsal to the inferior olivary nucleus. The cortico-spinal and cortico-pontile tracts were spared. In the *medulla oblongata* perivascular demyelination was found only in the reticular substance. The pyramids, restiform body and inferior olivary nuclei were free from lesions. Changes characteristic of retrograde degeneration were observed in the nerve cells of the *nucleus ambiguus* and of the lateral cuneate nuclei. The spinal cord was not available for examination.

DISCUSSION.

Study of the previous history of the patient failed to disclose any factor precipitating the condition described. The child had not been vaccinated against smallpox or immunized against diseases such as diphtheria, tetanus and whooping cough. Neither was there any history of recent respiratory infection, nor of an attack of any of the exanthemata. In this connexion it may be mentioned that the suggestion has been made that some cases of acute disseminated encephalomyelitis may occur subsequent to specific fevers in which the infection has been subclinical. For instance, it is readily conceivable that measles in the form of *"morbilli sine morbillis"* may be overlooked.

In the cases described earlier[17][7][9] the onset was relatively sudden. In the present case the onset was somewhat insidious, and clinically fell naturally into two stages. The first stage lasted approximately a month. It began with an initial attack of the disease on the cerebrum and was characterized by drowsiness and by changes in habits and personality. The parents noticed, not only that the child was unusually energetic, but that after exercise he became unduly tired. In the latter half of the first stage there were pains in the abdomen just below the costal margins, typical of "root" pains and suggestive of involvement of the spinal cord and/or its nerve roots at about the level of the tenth thoracic segment. At this period the child was noted to be unduly irritable and to feel the cold more than usual. It is of interest that, despite the symptoms described, the patient was still able to go to school up to within a few days of his admission to hospital.

The second stage was comparatively short, lasting five days. The drowsiness increased to coma, and spasms and rigidity of the arms and legs occurred. Similar symptoms were observed in Krabbe's case.[17] In addition both plantar reflexes were extensor in type and Kernig's sign was elicited. In the terminal stages there was also evidence of hypostatic pneumonia.

The cerebro-spinal fluid was examined on two occasions. On neither occasion was the pressure increased, but a slight pleocytosis was present, the white cells numbering about 30 per cubic millimetre; almost all of them were lymphocytes. In Greenfield's[7] case 17 cells were present per cubic millimetre, all of them mononuclear leucocytes. On the other hand, in the case described by Davison and Brock, polymorphonuclear leucocytes predominated among the 46 cells per cubic millimetre which were found. To judge by the experience of Marsden and Hurst,[9] in connexion with acute disseminated encephalomyelitis associated with smallpox, the type and number of cells present in the cerebro-spinal fluid are dependent on the stage of the disease at which the fluid is examined. In the present case the degree of pleocytosis conformed to the mild pia-arachnoid infiltration found at the post-mortem examination.

Pathological examination revealed a widespread distribution of lesions throughout the intracranial portions of the central nervous system. (It was unfortunate that the spinal cord could not be examined.) The pathological process was most evident in the white matter of the *centrum semiovale* and of moderate intensity elsewhere in the cerebrum and in the brain stem and cerebellum.

In all essential details the lesions were similar to those described in cases of idiopathic acute disseminated encephalomyelitis[1][7][9] and those occurring in association with vaccination, the exanthemata *et cetera*. There are, however, a number of features which require further discussion.

The occurrence of eosinophile polymorphonuclear leucocytes in the infiltrates is unusual. In none of the papers cited was I able to find any mention of them.

Compared with the findings in a case occurring subsequent to vaccination and described earlier,[11] the lipoid in the compound granular corpuscles was smaller in amount and the droplets were finer and stained reddish-orange, instead of bright red, with *Scharlach R* (Figure VI). The difference is probably due to the fact that the duration of the pathological process in the former case was about twice that of the present one.

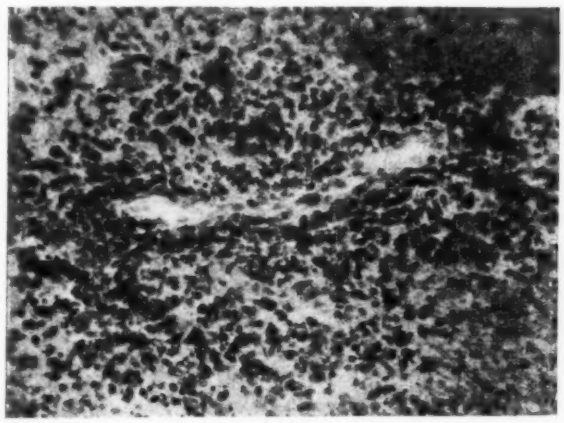

FIGURE VI.

Centrum semiovale. Perivascular and extraadventitial infiltration with lipoid-containing granular corpuscles. Frozen section. (*Scharlach R*; × 160.)

At first sight, in view of its character and of its virtual confinement to the *centrum semiovale*, the diffuse demyelination was attributed to œdema similar to that described by Greenfield[13] in association with intracranial tumours, and by Hurst[3] in connexion with acute hæmorrhagic leucoencephalitis. The changes, however, were too intense to be due entirely to œdema, and this suggested that there was a superadded attack by the causative agent.

In acute disseminated encephalomyelitis it is generally conceded that the perivascular lesions occur almost exclusively in association with venules and capillaries. In this regard the present case was similar. Where arterioles were affected, lesions were often limited to perivascular infiltration. If demyelination occurred, usually narrower rings were formed than those embracing veins. Dawson[30] concludes that in disseminated sclerosis and related conditions the causal agent is diffused through the walls of blood vessels, and this leads to solution of myelin together with a concomitant glial reaction. In disseminated encephalomyelitis following measles, Ferraro and Scheffer,[6] like Wohlwill,[4] subscribe to a similar hypothesis, but believe that the veins are especially affected owing to slowing of the blood stream and abnormal permeability of their walls. The findings in my case suggest that an important factor is the actual thickness of the vessel wall. On the basis of this suggestion it is assumed that, for the most part, the walls of the arterioles are thick enough to prevent the diffusion of the causal agent through them. Occasionally, however, the myelinoclastic factor succeeds in penetrating the walls of the arterioles, but usually in quantity insufficient to cause more than a narrow ring of demyelination.

Although it is concluded that the ætiological agent is distributed mainly by the vascular system, the fact should not be lost sight of that there is evidence to suggest that the cerebro-spinal fluid plays a role, probably a minor one, in the distribution of the causative factor.

Putnam[32] and his collaborators have been the chief protagonists of the theory, originally propounded by Ribbert[22] in 1882, that the basic mechanism in the demyelinating diseases is vascular obstruction. Putnam claims that in acute lesions, not only in disseminated encephalomyelitis but also in disseminated sclerosis, thrombi are found regularly in the vessels. The present case and the literature on the subject lend little support to Putnam's claim (see also Hurst and Cooke[22]). As far as disseminated sclerosis is concerned, Dawson[30] (see page 650 of his monograph) makes the following statement:

> In a few instances, especially in the lateral vessels of the cord and medulla, there have been found aggregations of white cells and the presence of fibrin, which have been taken as indications of intra-vital thrombosis, but nowhere has evidence been present of organization of such thrombi nor of alterations in the vessel walls in relation to them, nor have these been always in relation to sclerotic areas.

What is the relationship of acute disseminated encephalomyelitis to disseminated sclerosis? As Perdrau[12] points out, the primary feature of both diseases is the same, namely, perivascular demyelination. Putnam,[21] like Pette[20] and others, believes that the histopathology of the various demyelinating disorders is fundamentally similar, and that the differences in the lesions are due to variations in their intensity and location. In favour of such a view is the fact that cases[24][25][26][27] have been recorded, usually as of "acute disseminated sclerosis", the lesions in which are apparently transitional forms between acute disseminated encephalomyelitis and disseminated sclerosis.

The histopathological diagnosis between "acute disseminated encephalomyelitis" and "acute disseminated sclerosis" is sometimes very difficult. The differential points are discussed by Scheinker.[28] Of them, the most important facts in favour of a diagnosis of "acute disseminated encephalomyelitis" are that the lesions are exclusively perivascular and are uniform in their stage of development. Wohlwill[4] claims that in disseminated encephalomyelitis the whole length of the vessel is affected, while in desseminated sclerosis only a section is involved. Others have denied this claim. It has also been asserted that in acute disseminated encephalomyelitis the optic nerves are spared.

The results of animal inoculation experiments, like those of earlier investigators, were negative. There is thus little support for the view that disseminated encephalomyelitis may be due to the activation of a virus lying latent in the tissues, unless it is assumed that the virus is specific for man.

The only other explanation so far propounded to which credence can be given is the allergic hypothesis, but it is evident that much further experimental work is necessary. In this connexion it would be interesting to determine whether patients suffering from other phenomena of hypersensitivity, such as asthma, hay fever and urticaria, are more prone to demyelinating diseases than normal human beings.

SUMMARY.

Without any obvious exciting factor, a male child, aged seven years, manifested signs and symptoms of involvement of the nervous system characterized by drowsiness, changes in habits and personality, root pains and spasms and rigidity of the limbs. Death occurred in association with hypostatic pneumonia, about five weeks after the onset of the initial symptoms.

Histopathological examination disclosed acute disseminated encephalomyelitis with perivascular (extraadventitial) demyelination and its concomitant changes.

ACKNOWLEDGEMENTS.

I am indebted to Dr. E. Britten Jones for permission to publish the clinical record, and to Dr. E. B. Sims for the post-mortem material, which he generously placed at my disposal.

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Reviews.

LOCAL ANÆSTHESIA OF THE BRACHIAL PLEXUS.

R. R. MACINTOSH AND W. W. MUSHIN have written a little book of 56 pages which is explained by its title, "Local Anæsthesia: Brachial Plexus".[1] The authors state that the main disadvantage of brachial plexus block hitherto has been the uncertainty of success. There is no doubt about the truth of that, so long as the unmodified Kulenkampff method was practised. We imagine that many men have overcome the difficulty by devising for themselves a modification similar to that described in this book. We know that some have. If they have not, here is an excellent guide to show them. It takes up the surgical anatomy very carefully, as the first thing to learn. The authors are right. No one is qualified to carry out the technique of local anæsthesia for major surgery without an extensive and precise knowledge of surgical anatomy. That applies to much more than the special case of brachial plexus anæsthesia, and in this matter our authors have taken care to put the teaching of first things first.

The book contains 33 illustrations, which we can describe as luxuriously full and informative, and most artistically produced, giving welcome help to every intelligent beginner. The authors claim that the book largely "says it with pictures". The claim is justified, with an exception to be mentioned. This involves our one major criticism. There are a few small ones.

Our major criticism has to do with anatomy. Our authors have completely ignored the transverse cervical vein, which lies right in the field of action. When exposed in an open operation, and seen in its normal full condition, it is found to be a vessel of considerable size. It is true that needles usually pass by, and seldom puncture veins. But occasionally a vein is damaged, and there is some escape of blood through the needle if it is halted. When blood has escaped from the needle used for brachial plexus block, this is the vein that we ought to think of first, and to blame first. This bleeding is not a common occurrence, and not serious, but it does happen sometimes.

As the synonyms for procaine, we are given the names "Novocain" and "Planocaine", but we notice that the pharmacopœial synonym ethocaine is not mentioned. Yet some think it is a pity that this name is not preferred to procaine, because it is a much safer one.

We cannot agree with the recommendation to use amethocaine or "Nupercaine" for operations likely to exceed one hour, in preference to procaine. Procaine gives effective analgesia for well over two hours when it is combined with adrenaline, and though it is distinctly less effective after two and a half hours, some effect is still there. There can be very few operations on the arm that will take as long as two hours, even with a very slow surgeon. We have never seen one of anything like this length. Our authors do not mention the radical breast operation in connexion with brachial plexus block, though this block must be part of the technique. There is plenty of evidence that procaine is fully adequate in any breast operation.

We cannot agree at all with another of the recommendations, which is that in "shocked, frail, or toxic" patients the concentration must be markedly reduced. The authors advise reduction of procaine strength to 0·5% (or reduction of amethocaine or "Nupercaine" to 1 in 4,000). Nor do we agree that in these cases the adrenaline strength should not exceed 1 in 400,000. There is no warrant in experience for either of these reductions. We conclude that their recommendations are founded on *a priori* conjectures only. They are mistaken.

We do not think that the best instruction for preparing 2% solutions of procaine is to "dissolve 2 grams of procaine hydrochloride crystals in 100 c.c. saline". We hold that the ordinary man will not do it, whatever our authors do. It is better to advise him to use sterile tablets or capsules containing a given weight of the analgesic drug, and to dissolve them in the requisite quantity of 0·55% solution, which can be made by adding water to 100 parts of normal saline solution to bring it up to 163 parts. This is isotonic, which the solution the authors advise is not. Perhaps it is a minor matter for such relatively small quantities as fifty cubic centimetres. But why not do it, even if it is a minor matter? And as the English standard measuring vessel will be graduated in millilitres—near to, but not quite the same as, cubic centimetres—why not call them what they are, even if this is another minor matter?

Minor criticism is not necessarily hypercriticism.

[1] "Local Anæsthesia: Brachial Plexus", by R. R. Macintosh, M.A., M.D., F.R.C.S., D.A., and William W. Mushin, M.B., B.S., D.A., illustrated by Miss M. McLarty; 1944. Oxford: Blackwell Scientific Publications Limited. 7¼" × 5", pp. 59, with illustrations. Price: 10s. 6d. net.

Notes on Books, Current Journals and New Appliances.

BANDAGING AND FIRST AID.

THE appearance of a third edition of Sister Lois Oakes's book on bandaging and first aid is an indication of the need for this type of work.[1] The book has been translated into Spanish. Bandaging is taught in this book by pictures which are admirably suited to the purpose; one section deals with triangular bandages and another with roller bandages. There are short sections containing information for the first-aid worker on shock and hæmorrhage. In the section on first aid in fractures there is no insistence that the patient with fracture of the femur must not be moved until the Thomas splint arrives. This is a serious omission from an otherwise useful book.

[1] "Illustrations of Bandaging and First-Aid", compiled by Lois Oakes, S.R.N., D.N. (Leeds and London); Third Edition; 1944. Edinburgh: E. and S. Livingstone Limited. 8¼" × 5½", pp. 283, with many illustrations. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, JULY 7, 1945.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE REHABILITATION OF SERVICE MEDICAL OFFICERS IN THE UNITED STATES OF AMERICA.

EVERY member of the medical profession in Australia, whether he or she is engaged in civilian practice or is serving with one of the branches of His Majesty's forces, must by this time have some idea of the magnitude as well as the importance of the rehabilitation problem that will soon have to be faced in the world of medicine. The subject has been discussed by the Federal Council and also by the councils of the several Branches of the British Medical Association in Australia. On more than one occasion reference has been made to it in these columns. So far emphasis has been placed on the fact that service medical officers will be absorbed into the ranks of civilian practitioners only if those already in practice are prepared to treat the problem as if it was one that really concerned themselves. Those qualified to express an opinion hold that there will be little difficulty if practitioners throughout the Commonwealth try to find room for newcomers and are willing to treat them in a generous and fair-minded fashion. The alternative to a niggardly attitude and to exclusion tactics on the part of well-established practitioners is control of rehabilitation by a non-medical government department, with its possible unsolicited and inquisitive irruptions into other aspects of medical life. The existence of this alternative is real and we have been reminded of its existence more than once. To be spurred to action by its imminence would be to act from ignoble motives, but better that than complete apathy.

The willingness of civilian practitioners to open their ranks to medical officers discharged from the services and to help them to reestablish themselves is but one side of the problem. The other has to do with the wishes of the service officers themselves. No attempt must be made to push square pegs into round holes. Every service medical officer should be given an opportunity to state what type of practice he would like to undertake, and if possible he should be so placed that he will be able to do what he wants to do. No doubt a large number will ask for post-graduate instruction and training, and this should be looked on as only the first stage in their rehabilitation. From a number of sources *questionnaires* have been sent to Australian service medical officers and some replies have been received. The same procedure has been adopted in the United States of America, and an analysis of 21,029 replies to *questionnaires* has been made by Lieutenant-Colonel Harold C. Lueth, Surgeon General's Liaison Officer in the Medical Corps of the United States Army.[1] Conditions of practice in the United States are in many ways similar to those of the Commonwealth of Australia, and it has been thought that a short account of this analysis will be useful to Australian practitioners and to service medical officers. The former will learn something about the general requirements of service medical officers, and the latter may be led to take serious notice of *questionnaires* if they have not done so already.

The *questionnaire* was authorized by the Committee on Post-War Medical Service for distribution to each medical officer on duty with the Army, Navy, Public Health Service and Veterans' Administration. The Surgeons General of the Army, Navy and Public Health Service assisted with the distribution of the *questionnaires*. The 21,029 replies studied represented more than 35% of all medical officers on duty. Of the 21,029 medical officers who replied, 20·9% graduated in the period 1941-1943; 20·0% graduated in the period 1938-1940; 17·8% in the 1935-1937 period; 22·5% in 1930-1934; 15·8% in 1920-1929 and 3% before 1920. This means that more than 80% had graduated since 1930. It does not indicate that 80% of the officers in the services are of the later vintages, though in all probability they are; it does show that the younger men are most anxious to have post-graduate tuition. Nearly 40% of the 21,029 men had come to the services from private practice; 22% came directly from "internships"; nearly 10% came from "residencies"; and the remainder came from other types of practice. About 15% gave no answer to the question concerning their previous type of medical practice. Of the total number of officers, 92% had a licence to practise medicine—presumably analogous to Australian registration. There were 1,590 without licence; about 70% of these had graduated between 1941 and 1943, more than 20% of the unlicensed men were graduates of 1938-1940, and many of those who graduated before this were formerly engaged in work that did not require a licence and were in all probability returning to this work. There were no less than 3,922 medical officers, or 18·7% of the total, who stated that they did not want any future training. As might be expected, these comprised more of the older than of the younger graduates. About one-fifth of the group said that they wanted to undergo courses of training of less than six months' duration (these were called "short" courses); nearly 60% wanted to take "long" courses of further training in hospital or educational work. Requests for long courses covered periods of anything from six months up to two or three or more years. Requests for short courses included all the specialities. The greatest number of requests were for courses in internal medicine, and then in order of popularity came surgery, a "general review", obstetrics and gynæcology, pædiatrics, oto-laryngology, ophthalmology and so on. With those who

[1] *The Journal of the American Medical Association*, March 31 and April 21, 1945.

wanted long courses surgery was most in demand, and then in order came internal medicine, obstetrics and gynæcology, a "general review", psychiatry and neurology, pædiatrics, orthopædic surgery, ophthalmology, radiology, oto-laryngology and so on. It is interesting to note also that when the requests are considered in relation to the graduation groups of the officers, internal medicine held pride of place in every group among the short courses asked for, and surgery was the preference in every group of those who asked for long courses. One figure that will possibly cause some surprise is that 63% of the total group of 21,029 expressed a desire to become certified specialists. The figures concerning some of the courses of special study that are less in demand are not without interest. Only 270 officers asked for special work in pathology; of these, 60 wanted short courses and 210 long courses. It is significant that two-thirds of those who wanted long courses were younger graduates and that 178 of the 210 who asked for long courses were anxious to become certified specialists in the subject. In public health only 110 men asked for training; one-fourth of these asked for short courses and three-fourths for long courses. Of those who wanted long courses, 50 asked for one year of training, 17 wanted to be trained for two years, and six for three years. The subject of ophthalmology is of interest to Australians in view of the present shortage of younger ophthalmologists and the discussions that have taken place in this country regarding it. Those who asked for training in ophthalmology numbered 606; nearly one-third of this number wanted short courses, and the remainder long courses. Requests for short courses came mainly from the older groups. The requests for long courses were fairly evenly distributed among the graduation groups (except the oldest group), and 85% of them wished to be certified as specialists. No less than 73 from the first graduation group and 79 from the second group asked for long courses. The figures for oto-laryngology were very like those for ophthalmology. Some of the more restricted special subjects mentioned include hospital administration, industrial insurance, proctology, plastic surgery, neurological surgery and urology. When subjects such as these are mentioned, the wide range of subjects likely to attract medical officers in the services becomes apparent. Those in Australia who have to take an active part in the organization of rehabilitation will find more to interest them in Lueth's report than has been set out here; others, we hope, have been impressed with the greatness of the task and its urgency.

Current Comment.

D.D.T.

D.D.T., or dichloro-diphenyl-trichloroethane, has become established as one of the important drugs developed during this war. It was synthesized by a German chemist, Zeidler, as long ago as 1874, but it was not until 1939 that a Swiss chemical firm, Messrs. J. R. Geigy, A.G., of Basle, produced it for use as an insecticide against moths and plant lice. When it was subsequently shown to be very active against several other insects of medical and economic importance, production of the drug was expanded greatly, and today several tons are produced every day in both England and America.

Of the many recent papers on this drug, there are some of special medical interest. In February of this year, Professor P. A. Buxton in an address to the Royal Society of Tropical Medicine and Hygiene gave a general account of the drug, including some details of British work, hitherto unpublished.[1] Small samples of D.D.T. reached England towards the end of 1942 and were used with astonishing results. The substance proved to be lethal to lice and bed bugs at a tenth or less of the concentration required of the thiocyanates, which were among the most potent of insecticides then known. This discovery came at an opportune time, for the entry of Japan into the war and the occupation of Malaya had deprived Britain of half her supplies of pyrethrum and most of her supplies of derris; and, to make matters worse, the 1942 crop of pyrethrum in Kenya was poor. But D.D.T. proved more than a substitute for pyrethrum, and it became necessary to set about the production of D.D.T. on a large scale. This work was shared with America, where early trials had been proceeding parallel with the initial investigations in Britain.

D.D.T. is a white crystalline solid with a faint, pleasant smell. It is almost insoluble in water; it dissolves readily in various organic solvents, and is chemically stable; its volatility is low. It can be made by the interaction of anhydrous chloral and chlorobenzene in the presence of concentrated sulphuric acid.

For the control of body lice, D.D.T. can be impregnated into underwear, or diluted ten times with kaolin or an inert mineral and dusted onto the body. Clothing which has been impregnated with 1% to 2% by weight of D.D.T. will remain lethal to body lice for about eight weeks, even though it is washed weekly. This method of protection against body lice is of very great use for troops in the field. The other method has also had a large-scale trial during this war. In early 1944 there was a serious epidemic of typhus in Naples. D.D.T. powder was applied to the people by puffing it under the clothes, and the simplicity of this method made it possible to treat large numbers quickly; in January alone 1,300,000 civilians were treated. The outbreak was controlled within three weeks, and this was the first occasion known to medical science that an outbreak of typhus had been arrested in midwinter. It is difficult to over-estimate the importance of an effective weapon against epidemic typhus. During the war of 1914-1918 this disease killed 10,000 people in six months in Serbia; and soon afterwards it killed some three million Russians. Epidemic typhus broke out in Spain in 1941 and is a constant problem in certain South American States. At the same time though D.D.T. is certainly a valuable ally against epidemic typhus, it has so far not proved effective in combating mite-borne typhus, for example, scrub fever.

Adult mosquitoes are easily killed by sprays or aerosols containing D.D.T. Another useful method of killing mosquitoes is to coat the walls of houses with a film containing about 100 milligrammes of D.D.T. per square foot. This remains lethal for two to three months. Mosquito larvæ are extraordinarily susceptible to D.D.T. and can be killed by the incorporation of small amounts of the drug into anti-malarial oils. Other insects are killed by contact with D.D.T. It is, therefore, slower in action than pyrethrum, which has a "knock-down" effect. The usual symptoms of D.D.T. poisoning in insects are twitching of the legs and lack of coordination; death may be delayed.

There is no recorded case of human intoxication with D.D.T. in spite of the thousands of soldiers who have worn shirts impregnated with it and the large number of factory hands concerned in its manufacture. Nevertheless the search for possible toxic effects is very important, and in the circumstances, investigations on animals must continue to be carried out. A recent report of such experiments has been published by R. D. Lillie and M. I. Smith.[2] They showed, in work on rabbits, rats and cats, that in spite of the pronounced neurological symptoms, there were only minor histological abnormalities in the nervous system; but there were more striking degenerative changes in the liver.

Further details of the properties and uses of D.D.T. are given in two very readable reviews which recently appeared

[1] *The Lancet*, March 10, 1945, page 307.
[2] *Public Health Reports*, July 28, 1944, page 979.

in *Nature*—one written by Professor J. W. Munro[1] and the other by G. Lapage.[2] Professor Munro points out that under pressure of war D.D.T. has been tried on a lavish scale and that until the war ends it will not be possible to see D.D.T. and its competitors in proper perspective. Until the same exhaustive critical study as was given to D.D.T. is applied to other insecticides, it cannot be assumed that D.D.T. has ousted all competitors. Lapage gives some additional facts about the economic importance of D.D.T. It is lethal to head lice and crab lice as well as body lice. It is also effective against house flies, stable flies, ants, termites, certain cockroaches, and numerous plant pests. Among the proprietary preparations of D.D.T. are "Gesarol", available as a spray containing 5% D.D.T. and as a dust containing 3% D.D.T.; "Neocid", a dust containing 5% D.D.T.; and a spray, "De De Tane".

THE BIOSYNTHESIS OF VITAMINS.

IN the issue of February 3, 1945, attention was drawn in these columns to work by V. A. Najjar and his co-workers in which they showed that vitamin B_2 or riboflavin can be formed endogeneously in the human intestine. On that occasion reference was also made to previous observations which showed that thiamin can also be formed in the intestine of man. In view of some more recent work it will be useful to recall the main facts of the work done by Najjar and E. Holt on thiamin.[3] These investigators fed a diet low in thiamin to nine young men for eighteen months. As the diet consisted only of casein, "Crisco", dextri-maltose, a mineral mixture and vitamins, it is obvious that the subjects showed a rare degree of enthusiasm and cooperation. As the experiment proceeded it was found that the expected signs of thiamin deficiency failed to appear, and it was decided to omit thiamin entirely from the diet. In the course of a further seven weeks, four subjects remained quite healthy and the other five showed such expected symptoms as neuritis, œdema, anorexia and vomiting. It was then found that an abundance of free thiamin was present in the fæces of those subjects who were free of symptoms. It seemed highly probable that this thiamin was formed in the intestine by bacterial action, and evidence strongly supporting this was obtained when the excretion dropped to zero after the administration of succinylsulphathiazole. However, the further problem remained, whether the thiamin formed in the intestine was produced in a region where it could be absorbed. This was tested by the giving of retention enemas containing thiamin, and the subsequent observation of the effect on the elimination of thiamin in the urine. It was found that the vitamin introduced into the large intestine in this way produced a pronounced rise in the urinary thiamin.

More recently two papers have been published in which the conclusions of Najjar and Holt are not entirely substantiated. B. Alexander and G. Landwehr[4] agree that thiamin and its combined form, cocarboxylase, are synthesized in the human intestine; but they contend that the vitamin remains in the bodies of bacteria and is excreted in the fæces. Their evidence for this is that if an aqueous suspension of fæces is treated by Seitz filtration, the thiamin does not pass through the filter, but remains with the bacteria. Moreover, when a subject is given an enema containing twice the amount of free and combined thiamin that he normally excretes per day, there is no increased excretion of thiamin in the urine, and all of the administered thiamin can be recovered next day in the fæces. Alexander and Landwehr point out that when Najjar and Holt performed a similar experiment an abnormally large dose of thiamin was given, and it was all in the form of free thiamin. The cocarboxylase content of the fæces is about five to twenty times that of the free thiamin, and cocarboxylase is not absorbed even in the small intestine until the phosphate portion has been split off. It is doubtful whether the phosphorylating enzymes necessary for this exist in the large intestine. The work of A. Williamson and H. T. Parsons[1] is concerned with the effect of a high fibre diet on the excretion of thiamin in the fæces. They showed that subjects on these diets excreted an increased amount of thiamin in the fæces, and that these increases were due to stimulation of synthesis by intestinal bacteria. In such circumstances one might have expected an increase in the urinary excretion of thiamin, if the thiamin formed in the intestine could be absorbed. Such an increase, however, was not consistently obtained. It is clear that more work on this fascinating subject is needed.

[1] *Nature*, September 16, 1944, page 352.
[2] *Nature*, November 11, 1944, page 600.
[3] *The Journal of the American Medical Association*, Volume CXXIII, 1943, page 683.
[4] B. Alexander and G. Landwehr: "The Role of Fecal Thiamine and Cocarboxylase in Human Nutrition", *Science*, Volume CI, March 2, 1945, page 229.

VITAMIN B IMBALANCE THROUGH EXCESS OF ONE FACTOR.

AN important investigation on vitamin *B* imbalance has recently been completed in the Rowett Research Institute of Aberdeen and published in the *British Medical Journal* of March 31, 1945. Put briefly, the outstanding discovery here announced is that there is a subtle interaction and balance of function between the members of the *B* group so that excess of one may produce unmistakable indications of deficiency in another, or if deficiency in this has been present before, then this deficiency will be intensified. When, for example, in animal experiments thiamin was administered above the body's needs the very obvious signs of inadequate pyridoxine developed. This research has cleared up many puzzling facts described by other investigators and by clinicians. In the human patient large doses of nicotinic acid given to cure pellagra caused signs and symptoms associated with insufficient pyridoxine. Several clinicians, quoted in this article, have described untoward effects following medication with thiamin, and these bore striking resemblance to conditions arising from lack of one or two or more other members of the *B* group. The author, Dr. Marion B. Richards, utters a warning concerning the treatment of a deficiency syndrome by one *B* vitamin alone. "Danger may lie, however, in the present-day tendency to prescribe vitamin B_1 somewhat indiscriminately as a dietary adjunct and to give large doses of B_1 orally or by injection in the treatment of various diseases. It is precisely in such cases, in which the patient is probably on invalid diet that is liable to be unbalanced, that a large excess of B_1 may entail unexpected and dangerous results." There is something in these facts which recalls early physiological work on the mineral composition of perfusion fluids for excised organs, particularly the heart. It was noted, for example, that raising the concentration of magnesium, not only produced magnesium depression, but also derangements suggestive of calcium lack. When the calcium content was raised to maintain the old ratio, an immediate betterment resulted. Apparently in the *B* vitamins the ratios are as important as the absolute amounts.

At the present time exceptional emphasis is being placed on thiamin and not a little uncritical enthusiasm is manifest. We are told that sugar is so much "dead weight", as it contains no thiamin, and so its metabolism is dependent on the thiamin obtained from other sources. This is no doubt true, but those who in consequence roundly condemn the sweet tooth of the Australian soldier in the tropics should be cautious, especially if their opinions are translated into authoritative actions. Lord Horder's warning that we should not be dogmatic in nutritional matters unless we know all the facts, and we often do not, still remains valid. Anyhow, Dr. Marion Richards brings us back to the view that the naturally occurring vitamins are safer, as they are usually multiple and possess the correct ratios.

[1] A. Williamson and H. T. Parsons: "Some Factors Influencing the Fæcal Elimination of Thiamine by Human Subjects", *The Journal of Nutrition*, Volume XXIX, January 10, 1945, page 51.

Abstracts from Medical Literature.

GYNÆCOLOGY.

Biological Changes in Squamous Epithelium Transplanted to the Pelvic Connective Tissue.

FRANK E. WHITACRE AND Y. Y. WANG (*Surgery, Gynecology and Obstetrics*, August, 1944) have operated on two patients born without a vagina. They split up a space between the bladder and the rectum and inserted a skin graft from the inner surface of the thigh applied to a "Pyrex" glass mould. This thin layer of epithelium grew rapidly, acquired ridges or rugæ, and a pink-blue colour. Within six months it has assumed to a remarkable degree the appearance of a normal vagina. Histological examinations were made six months after the operation, when it was found that the skin that had been implanted had taken on all the characteristics of normal vaginal lining, showing the three principal layers of Dierks; that is, the basal layer, functional layer and zone of cornification, with a layer of glycogen in the third layer. Œstrogenic substance was not given to any of the patients until all the examinations were made. The vaginal secretions became normally acid within six months. The authors concluded that the changes were not due to control by œstrogenic tissue, but were apparently due to a gross stimulating substance which was probably derived from the surrounding connective tissue. Within six months of the original operation the cellular structure, acidity and bacterial flora of the artificial vagina were almost identical with those of a normal vagina.

Benign Menopausal Bleeding.

WALTER J. REICH, HELEN BUTTON AND MITCHELL J. NECHTOW (*Western Journal of Surgery, Obstetrics and Gynecology*, November, 1944), working in the gynæcological section of the Cook County Hospital, have reported a series of thirty cases of benign menopausal bleeding. This condition is one of very considerable importance, especially when it is remembered that bleeding at or after the menopause often is due to cancer. The authors report a series of cases in which menopausal bleeding was definitely proven not to be due to malignant disease; they exclude cases in which bleeding occurred from the bladder, ureter, kidney and gastro-intestinal tract. They classify the conditions as follows: Post-menopausal vaginitis and cervicitis which give rise to contact bleeding during and after coitus, and which usually result from the climacteric. Cervical "erosions", which can only be distinguished from cancer by a biopsy. Cervical polypi, which occur frequently in cervical stumps left after supracervical hysterectomy. Papillomata of the vagina, which are usually flattened papules. *Trichomonas vaginalis*, which shows the usual organisms under the microscope. Urethral caruncles. Endometrial polyposis in the vagina. Submucous fibroids. Degeneration of intramural fibroids, either hyaline or cystic. *Fibrosis uteri* occurring in a fairly large uterus. Yeast vaginitis due to the presence of yeast organisms in the vagina. "Stilbœstrol bleeding", due to taking an excessive amount of stilbœstrol given for post-menopausal conditions. Granulosa cell tumour, which often causes the periods to return apparently in a normal way, and which requires laparotomy with removal of the tumour or hysterectomy with bilateral salpingo-oophorectomy. Theca cell tumour. Gaertner duct cyst, which occurs frequently high up on the lateral portion of the vaginal vault. Prolapse of the fimbriated end of the uterine tube, following a vaginal hysterectomy. Hypertension or uterine epistaxis, which requires medical treatment. Post-irradiation tissue changes caused by the use of radium or deep X-ray therapy, which requires soothing douches of lactic acid and menthol. Changes in climate from a cold to tropical area causing bleeding which is probably due to some vaso-endocrine disturbance. *Procidentia uteri* with decubitus ulcer of the cervix. Endometriosis and *adenomyosis uteri*. Pelvic inflammatory processes, particularly tuberculosis. Benign hyperplasia of endocervical epithelium. Blood vessels coursing over Nabothian cysts. (All these conditions can be diagnosed by examination through the speculum.) Chancre of the urethra occurring occasionally and having to be diagnosed by dark field examination. Chancre of the *labia minora*. Pyometra after the menopause due to the senile cervix becoming closed, or almost closed, with a low grade endocervicitis and endometritis behind it. Metallic foreign bodies left in the uterus for purposes of contraception. Ulceration of the vagina from an improperly fitting diaphragm for contraception. Varicose veins of the vagina. Blood dyscrasias. The authors quote a series of cases to illustrate these conditions, but found the old dictum true that all conditions causing menopausal bleeding are malignant until proven otherwise.

Full-Time Intraligamentous Ectopic Gestation.

LILY G. SELLIAH AND W. C. OSMAN HILL (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1945) report a rare case of full-time anterior subperitoneal intraligamentous pregnancy. It was discovered in one of the bodies sent to the dissecting room of the department of anatomy in the University of Ceylon. The body was that of a patient twenty-nine years of age, whereas it had the appearance of a woman of nine years of age. It was her second pregnancy. She had had a normal delivery eight years previously. On abdominal palpation a hard mass, thought at first to be a fœtal head, was felt high up to the left of the middle line and the fœtal back to the right side. On opening the abdomen a large thin-walled sac was found to occupy the centre and right of the pelvis and lower part of the abdomen. The thin wall of the sac was accidentally penetrated by the knife and revealed the back of a full-time child; its buttocks were disposed upwards and to the right and its head was later discovered well down in the pelvis in the right occipito-anterior position. Amniotic fluid was absent; the membranes were lying on the surface of the child. The anterior peritoneal wall of the gestation sac was reflected along the inguinal region on the posterior aspect of the anterior abdominal wall. The stretched *ligamentum teres* extended across the sac towards the internal abdominal ring, the caecum and the appendix were closely adherent to the top of the sac on its extreme right, the right Fallopian tube was found along the upper end of the sac, the right ovary was normal, and lay to the right of the tube and above the sac. The uterus was displaced to the left and upwards, and enlarged to the size of the fœtal head and hard to the touch. This was the object which had given the impression of a fœtal head on abdominal palpation. The uterus was six inches long and four inches broad, and one and a half inches thick in the wall. The left tube was normal in length and compressed, the right tube was stretched to four and a half inches. The placenta was found attached to the medial half of the upper part of the anterior wall of the sac, passing thence on to the right lateral wall of the body of the uterus between the two layers of the broad ligament. Its upper margin reached to the Fallopian tube, but whether or not there was any original placental attachment, or alternatively whether this was due to secondary extension, could not be definitely stated. The area in contact with the uterine wall included the portion of the latter that had been eroded, the erosion producing a rupture of the uterus. The child was a well-developed full-time male, weighing three pounds one and a half ounces. The authors discuss the rarity of the condition and conclude that the condition was one of true mesometral pregnancy. The pregnancy had proceeded to term and the erosive action of the placenta had produced a secondary opening into the birth canal. Lack of expulsive power around the wall of the gestation sac had resulted in the infection of the fœtal head in the maternal pelvis. The mother had died not so much from hæmorrhage as from shock.

Relationship of Glycogen to Problems of Sterility and Ovular Life.

EDWARD C. HUGHES (*American Journal of Obstetrics and Gynecology*, January, 1945) reports the results of a study of endometrial and ovular tissue from a chemical point of view. He states that all the cells of the female reproductive system contain a supply of glycogen. He has found that the amount present varies with different phases of the menstrual cycle, and there is a corresponding variation in the glycogenolytic enzyme occurring along with the glycogen. He maintains that the purpose of this glycogen is to provide the ovum with nourishment during its migratory and early post-nidatory phases. In the cells of the endometrial glands a definite chemical cycle can be traced. It commences with the extraction of glucose from the circulating blood in the early follicular phase and its conversion and storage as glycogen granules. During the progestational phase, gradually increasing amounts of this glycogen are broken down by enzymatic action to the readily assimilable form of glucose. Applying this to his studies of sterility, the author found that in many women, who appeared to be normal in every other way, glycogen and enzyme concentrations were below normal when an endometrial biopsy was made about the twenty-sixth day of the cycle. This condition was found in some

patients with a fairly normal progestational pattern. The author suggests that this fact may be the cause of sterility in these cases or of spontaneous abortion if conception does occur. He believes that the glycogen metabolism is probably influenced by the ovarian hormones, and that the treatment of these patients with ovarian preparations has a good chance of success. The treatment should be instituted before pregnancy and continued in the early days of gestation. In conclusion, the author suggests that although his studies may not be conclusive, they indicate a new line of research in uncovering cases of sterility and abortion which do not fit into any of the already known classes.

Ovarian Fibroma with Ascites and Hydrothorax (Meigs's Syndrome).

R. H. GARDINER AND V. LLOYD-HART, of the Royal Bucks Hospital, Aylesbury (*The Lancet*, October 14, 1944), report a series of four cases of benign ovarian fibroma associated with ascites and hydrothorax. In reviewing the literature, they find 38 cases of a similar type. The symptoms are mainly ascites with hydrothorax of varying degree. The hydrothorax may be due to small congenital openings in the diaphragm which allow direct passage of fluid from the abdomen, or the fluid may enter the pleural cavity via the diaphragmatic lymphatics, or there is mechanical obstruction of the venous or lymphatic outflow of the thorax. The authors consider that it is mainly due to mechanical obstruction to the venous return of the heart. The main point is that these are frequently diagnosed as malignant ovarian conditions. Fibroma of the ovary should be considered in all women complaining of shortness of breath or pain in the chest with enlargement of the abdomen and pressure symptoms; while many of the fibromata are malignant, certain cases belong to this simple group and clear up completely with removal of the fibroma of the ovary.

Pelvic Lymphadenectomy in the Treatment of Cervical Cancer.

DANIEL G. MORTON (*American Journal of Obstetrics and Gynecology*, January, 1945) reports 65 cases of carcinoma of the cervix in which treatment consisted of a combination of X-ray therapy and pelvic lymphadenectomy. The basis for this treatment is the belief of many surgeons that glandular metastases are less responsive to irradiation than the primary cervical carcinoma. The cases suitable for this combined treatment are those in which regional lymph node metastases exist, even when the local growth is early enough to permit destruction by irradiation. One of the great difficulties in deciding the indications for lymphadenectomy is the uncertainty of present-day knowledge of the incidence of glandular involvement, especially in relation to the degree of advancement of the local growth. The glands involved first are the so-called first-stage glands, comprising the obturator, hypogastric, ureteral and sacral groups. The second-stage glands, such as the inguinal and the lumbar groups, are usually involved only late in the disease. The author selected for operation those cases in which the immediate reaction of the primary lesion to irradiation was good.

OBSTETRICS.

Maternal Obstetrical Paralysis.

JOHN E. A. O'CONNELL (*Surgery, Gynecology and Obstetrics*, October, 1944) has investigated the occurrence of those conditions of neuritis of pregnancy and the puerperium which affect one or both lower limbs. The condition takes the form of a so-called peripheral neuritis and may arise in pregnancy or follow labour. Investigation of a series of these cases showed that the condition is more common than is generally appreciated. X-ray examination of a series of patients showed that in a proportion of cases the disability was the result of protrusion of a lumbar intervertebral disk. It is well known that there is a softening of the tissues in the intervertebral and pelvic joints in the late stages of pregnancy which cause the normal stress and strain of labour when the patient is in the lithotomy position to bring undue force to bear on these intervertebral disks. The disk may protrude into the vertebral canal and cause pressure on the cord. There seems to be no doubt that the severe objective neurological disturbance which not infrequently occurs when a disk protrudes may be accounted for by the large size and rapid development of the protrusion with the consequent severe involvement of nervous tissue. Patients may be severely incapacitated for a considerable time as the result of this pressure on the cord. They will require a considerable amount of treatment with rest, splinting and electrical stimulation for the muscles. Operative interference may have to be considered. In any such case it is advisable to get the opinion of an orthopædic surgeon.

Cervical Pregnancy.

CLIFFORD L. FEARL (*Western Journal of Surgery, Obstetrics and Gynecology*, March, 1945) reports a case of cervical pregnancy. He states that a search of the records in two Portland hospitals, covering a period of thirteen years and including 26,345 deliveries, failed to reveal another instance of the condition. Pregnancy in the cervical canal is regarded as an ectopic pregnancy. Speed and Rock in a recent study have stated that if the speed of descent of the fertilized ovum is such that it is found in the cervical canal when it has reached the proper stage of growth for becoming imbedded, then the pregnancy may develop in this site. The probable reason for the infrequency of cervical pregnancy is the unfavourable nature of the tissue for the early nourishment of the developing ovum. Rubin, in 1911, in a report of seven proven cases in the literature at that time, stated four criteria which must be fulfilled if a certain diagnosis of cervical pregnancy is to be made: (i) there must be cervical glands opposite the placental attachment; (ii) the attachment of placenta to cervix must be intimate; (iii) the whole or a portion of the placenta must be either below the entrance of the uterine vessels or below the peritoneal reflection on the anterior and posterior surfaces of the uterus; (iv) fœtal elements must not be present in the *corpus uteri*. The mortality rate is high. Hæmorrhage due to rupture of such an ectopic pregnancy is alarming and difficult to control. Sepsis may bring about a fatal termination. In the author's case, the patient, a janitress, aged forty years, complained of inability to pass urine, of four days' duration. Examination disclosed a cervical pregnancy. Operation was performed, and the uterus was emptied. The fœtus was 1·5 centimetres in length; it was just inside the external os, and moderately adherent placental tissue was also present. Microscopic examination of the tissue failed to reveal any chorionic villi in immediate association with cervical glands. The patient had noticed a little nausea in recent weeks, with a sense of fullness and tingling in her breasts. She had decided that she was not pregnant, and had attributed her two months' absence of menstruation to an early menopause. Her past obstetrical experience included a number of induced abortions and no full-term pregnancies.

The Significance of Signs and Symptoms in Toxæmia of Pregnancy.

FRANCIS J. BROWNE (*Edinburgh Medical Journal*, November-December, 1944) gives a *résumé* of the theories propounded up to the present time to explain the underlying mechanisms of œdema, hypertension and albuminuria in toxæmia of pregnancy. With regard to œdema, he states that in some cases it may be the cause of eclamptic convulsions by bringing about in the brain anæmia and anoxia of the nerve cells. In other cases, however, the same effect may be produced by arteriolar spasm and hypertension, even in the absence of œdema. The author denies the importance of an antidiuretic pituitary secretion in the causation of œdema of pregnancy. He also refutes the theory that hypoproteinæmia is an important causative factor, pointing out that the serum proteins are usually well above the critical level in preeclamptic toxæmia. A theory which obtains some support from the author involves the action of the hormones of the adrenal cortex. These hormones are closely related chemically to the female sex hormones, and when injected into humans can cause sodium retention and œdema. Discussing hypertension as it occurs in pregnant women, the author points out that only essential hypertension can be caused by constriction of the renal arterioles. In preeclamptic toxæmia, it has been demonstrated that an appreciable dilatation of the renal arterioles occurs. Experiments, in which a pressor substance is injected into normal non-pregnant and pregnant women and into patients with preeclamptic toxæmia, have shown that women in the toxæmic group are more sensitive and have a greater rise in blood pressure than normal women. This sensitivity to pressor substances appears to be acquired in some way during the course of the pregnancy, and may be of importance in explaining the tendency of pregnant women to develop hypertension. Thirdly, with regard to albuminuria, the author states that a good deal is known, but probably not everything. In cases of essential hypertension during pregnancy, it is known that the albuminuria is due to arteriolar spasm causing anoxia and abnormal permeability of the glomerular capillaries. He suggests that this may not be the sole cause of albuminuria in preeclamptic toxæmia, especially if it is proved that hypertension does not always precede albuminuria in these cases.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal Alexandra Hospital for Children on April 17, 1945. The meeting took the form of a series of clinical demonstrations by the members of the honorary medical staff of the hospital. Part of this report was published in the issue of June 16, 1945.

Cerebral Abscess.

DR. T. Y. NELSON showed a male patient, aged nine years, who had been admitted to hospital on October 24, 1944. The child had been vomiting nearly every day for five or six weeks. Periods of drowsiness had been observed, with slowing of the pulse, and in the last two weeks a squint had developed. He complained of constant headache across the top of the head, and of pain behind the left eye.

Examination showed the child to be thin and drowsy, with evidence of dehydration. He continued to vomit after his admission to hospital. Examination of the cranial nerves revealed intense papilloedema of each optic nerve up to five diopters, with enlargement of the veins and some scattered hæmorrhages. Partial ptosis of the left eye was seen. The pupils were equal and reacted to light. No involvement of the fifth nerve could be detected, but the slight squint which was present indicated pressure on the left sixth nerve. Some paresis of the right side of the face could be seen. No involvement of the other cranial nerves could be detected. Extreme muscular hypotonia was present, and although it was difficult to obtain much cooperation owing to the drowsy condition present, it was thought that some ataxia of the left arm was present; but there was no nystagmus. The abdominal reflexes were weak on the right side, and the knee and ankle jerks were absent. X-ray examination of the skull revealed slight widening of the coronal suture. Lumbar puncture was performed, and showed the pressure to be 250 millimetres of cerebro-spinal fluid. A small amount of fluid was removed for examination; it was found to contain one mononuclear cell per cubic millimetre. The test for globulin produced a positive reaction, the chloride content of the fluid was 720 milligrammes per 100 cubic centimetres, and the protein content was estimated at fifty milligrammes *per centum*. The presence of some cerebellar signs suggested the possibility of a posterior fossa tumour, and it was thought that the sixth and seventh nerve involvement might be false localizing signs and arrangements were made to perform ventriculography. However, a small fluctuant swelling developed over the left frontal bone and rapidly assumed the appearance of a "Pott's puffy tumour". An incision was made over the affected area and pus was found under the periosteum. The softened, infected bone was nibbled away, a circular area three centimetres in diameter being exposed, and the dura was coagulated around the periphery of the opening. A brain needle encountered an abscess cavity about one centimetre below the surface. The dura was opened and the abscess was "uncapped" by removal of the overlying cortex with the diathermy loop, and the abscess cavity was opened and drained. A glove finger packed with gauze was left in the cavity.

The patient's general condition improved after operation and continued to be satisfactory. Cerebral hernia developed at the operation site, and the drain was forced out. Lumbar puncture on November 21 showed the cerebro-spinal fluid to be under a pressure of 400 millimetres, and thereafter lumbar puncture was performed each day to reduce the pressure. On November 24 the wound was dressed in the operating theatre, and a brain needle introduced to explore the possibility of an undrained abscess encountered a diverticulum of the ventricle which had been forced up into the herniated brain. For about twelve days afterwards cerebro-spinal fluid continued to drain from the hernia, and it continued to do so intermittently until December 19, when the fistula closed and remained closed. Infection was controlled by sulphadiazine and penicillin, and the hernia gradually subsided, lumbar puncture being performed frequently until January 15, 1945. Dr. Nelson said that the hernia had now healed, and the child was left with secondary optic atrophy. He could see well enough to get about, but could not read. Arrangements had been made for him to attend the school for the blind.

Patent Ductus Arteriosus.

Dr. Nelson next showed a male child, aged three and a half years, who had been admitted to hospital on July 27, 1944. The mother had suffered from German measles in the third month of pregnancy, and the child's physical development had been retarded. There was no indication of congenital cataract. The child made no attempts to speak, but his intelligence was fair. He was under the care of Dr. Donald Vickery, and was referred for operation on a patent *ductus arteriosus*. There was no history of cyanosis. Physical examination showed that the apex beat was in the fifth left intercostal space two and a half inches from the mid-line, and a thrill could be felt over the precordium. A loud "machinery" murmur was heard all over the precordium and also posteriorly on the left side, the maximum intensity being over the second left intercostal space. X-ray examination revealed enlargement of both ventricles, and of the pulmonary arc, which the radiologist, Dr. K. Voss, considered suggestive of a patent *ductus arteriosus* and a patent interventricular septum. The blood pressure was 85 millimetres of mercury, systolic, and 40 millimetres, diastolic.

Operation was undertaken on August 1. Anæsthesia was maintained by nitrous oxide and oxygen, with a small amount of ether, given by Dr. Morgan through a closed system. Transfusion of blood was commenced before the operation and maintained by the "slow drip" method. The thorax was opened through the second intercostal space; the *pectoralis major* was split to provide access, and the costal cartilages of the second and third ribs were divided to enable the space to be opened out. Dissection of the mediastinum was carried out as described by Gross, the phrenic nerve being easily identified, and the ductus was displayed and ligated at each end with number 4 silk sutures. The size of the patent ductus was approximately one centimetre in length and in diameter. The thrill was abolished immediately after ligature was completed. There was also an immediate rise in systolic blood pressure to 120 millimetres of mercury, but no alteration in the diastolic pressure at this stage. The mediastinal pleura was sutured and the chest was closed without drainage. Satisfactory lung expansion occurred, and no pleural effusion developed. Examination of the heart immediately after the operation showed that no murmur was present, and blood pressure readings taken after the patient's return to bed showed that the systolic pressure was 120 millimetres of mercury and the diastolic pressure 70 millimetres. These readings were constant for a few days, but at the end of a week they had fallen to 95 millimetres and 70 millimetres respectively, and these levels were maintained until the patient's discharge from hospital. X-ray examination on August 11 showed that the lung was fully expanded, and the heart shadow had lost its former globular appearance.

The child was examined again on December 9 by the committee appointed by the Director-General of Public Health of New South Wales to investigate congenital defects following maternal rubella during pregnancy. The committee reported having found no evidence of cardiac enlargement; a short systolic murmur was present, best heard in the third left intercostal space. The committee also found that the child's concentration was good, that he was able to say a few single words, and that he could hear a door bell, an aeroplane and sharp noises. The general impression was that this child was capable of being taught a trade so as to earn his own living.

On December 19 a further X-ray examination revealed little alteration; the heart was slightly enlarged. The appearances suggested a septal defect. The child was tested by experts from the Deaf, Dumb and Blind Institution; they reported that he was playful and natural and made intelligent use of his eyes, that he had good practical problem-solving ability, and that the mother stated that he was doing some lip reading. The prospects for a normal deaf school education were considered very good.

Dr. Nelson said that this case raised the question of what was the optimum age at which ligature of a patent *ductus arteriosus* should be carried out. It had been suggested that spontaneous closure might occur in the first few years of life, but by the age of four years the condition should be well established, and the operation presented no great technical difficulty at that age, and was probably more easily performed than on an adult patient. It was evident that this was an operation essentially indicated in childhood, when it should be regarded as a prophylactic measure to close an arterio-venous shunt. Difficulty lay in the uncertainty of diagnosis, and in the uncertainty of maintaining closure; but with increasing experience the latter problem should be solved.

Subdural Hæmatoma.

Dr. Nelson's next patient was a baby, aged five days, who had been admitted to hospital on December 27, 1944, from

the Women's Hospital. The baby weighed seven pounds fifteen ounces, and the birth was described as having required a difficult instrumental delivery. A typical "pond" fracture was seen in the right parietal region. The particular point of interest was that twitching of both arms and legs was noted, and was more pronounced on the left side. As this was an unusual feature of the ordinary depressed fracture of infants, the possibility of a subdural hæmatoma was considered. The temperature varied between 102° and 100° F. Double subdural aspiration was performed on December 29 through the coronal suture lateral to the fontanelle, and on the right side about one cubic centimetre of dark blood-stained fluid was removed. From the left side about 0·5 cubic centimetre of fluid was removed; it was much paler in colour. The baby's condition improved, the temperature gradually returned to normal and remained so, and no further twitching occurred. On January 2, 1945, a small burr hole was made in the posterior parietal region and the fracture was elevated. The dura was opened, to find out the extent of the clot, but no abnormality could be seen in this area. Subdural aspiration was then again performed on the right side, and only a small amount of fluid was obtained, much paler in colour than on the first occasion. Progress was satisfactory. The baby gained weight normally, and on January 23 a final aspiration was performed, at which only a few drops of clear fluid were obtained from each side.

Dr. Nelson said that the case was of interest, in that it was not associated with the massive clot extending over a large area of the cerebrum which was usually described in such cases. The small collection of blood found anteriorly was sufficient to cause cortical irritation, and a decided change in the baby's condition was noted when it was removed.

Subarachnoid Cyst.

Dr. Nelson then showed a male patient, aged seven and a half years, who had been admitted to hospital on November 13, 1944. He had been in hospital in 1943 with a history of "turns" of three months' duration, several occurring each day without loss of consciousness. At times the left hand would twitch. No gross changes were found on examination of the central nervous system, but some incoordination of the left hand was noted. A plain X-ray film of the skull revealed a series of markings in the right parietal region, which were shown by stereoscopic examination to be diploic veins. Encephalography was performed, and a large shadow was seen in the right side which was thought to represent a cavity in the cerebral substance. The child was given "Dilantin", 0·5 grain twice a day, and allowed to go home.

Observation by the follow-up department showed that he had no more seizures, but was mischievous and at times irritable. He appeared to be of subnormal mentality. He was readmitted to hospital and craniotomy was performed on November 14, 1944. A bone flap was turned down in the temporal region. The dura was opened, and immediately beneath the dura a collection of fluid was seen which had the appearance of cerebro-spinal fluid. About two ounces of this fluid were sucked out, and the surface of the temporal lobe could then be seen. In the depths of the wound a small nodule of tissue, measuring about one centimetre in all directions, was seen to be attached by a pedicle apparently to the surface of the brain. This had floated freely in the fluid-filled cavity. A silver clip was applied to the pedicle and the tissue was removed for examination. The bone flap was replaced and the wound was closed. Post-operative recovery was slow, owing to meningeal infection. On the third day the temperature was elevated, and by the sixth day the child had become drowsy with periods of irritability and wild delirium. Neck rigidity developed, and lumbar puncture yielded turbid fluid, which contained 1,500 polymorphonuclear cells per cubic millimetre and from which a culture of hæmolytic streptococci and *Staphylococcus albus* was obtained. This infection was controlled by sulphadiazine, and later, because of some discharge from the wound, penicillin was given. Repeated examinations of the cerebro-spinal fluid over the next six weeks revealed a gradual decrease in the cell content and in the protein level until on January 19, 1945, the boy was considered fit for discharge from hospital. Dr. Nelson said that he had remained well, took no sedatives and was improved mentally. Microscopic examination of the tissue removed at operation was carried out by Dr. H. K. Reye, who reported that it was composed of an outer layer of choroid plexus, which was lacking over only a small portion of the circumference, and a cone of fibrillary astrocytes containing many cysts filled with a pink coagulum; occasional rounded discrete areas of calcification were to be seen. Dr. Nelson said that the condition was called a subarachnoid cyst for want of a better name. It did not belong to the class of case described as "cystic arachnoiditis" following an inflammatory reaction, but apparently corresponded to an abnormally placed "cistern" due to the presence of an aberrant piece of choroid plexus.

Septicæmia Treated by Penicillin.

The next patient shown by Dr. Nelson was a boy, aged eight years, who had been admitted to hospital on March 8, 1945. The child had been ill only since the previous night, the main symptoms being vomiting and abdominal pain. He was admitted to hospital under suspicion of poliomyelitis.

Examination revealed some stiffness of the spine, and the muscles of the back, the abdominal wall and both calves were tender to pressure. No paralysis was evident, and no neck rigidity. The temperature was 103° F. Lumbar puncture was performed, and the cerebro-spinal fluid was clear, and contained no cells; the chemical investigation gave normal findings. On the following day the child's condition had deteriorated. The temperature remained 103° F., he was vomiting frequently, the tongue was dry and he was irrational. The administration of saline solution intravenously was commenced and penicillin therapy was started, 15,000 units being injected intramuscularly every three hours. Examination of the blood showed that the leucocytes numbered 26,600 per cubic millimetre, 85% being polymorphonuclear cells.

At this stage a surgical consultation was arranged, and examination showed that although the abdominal muscles were tender to palpation, there was no rigidity or evidence of peritonitis. Examination of the limbs showed that the lower end of the left femur was acutely tender on deep pressure; this suggested a commencing bone infection in this area. Dulness at the base of the left lung was noted, and by the following day well pronounced breathing was present.

Blood was taken for cultural examination on March 9, but failed to yield any organisms. Dr. Nelson remarked that as this test was not carried out until after the penicillin injections were given, it was likely that the penicillin inhibited the growth of organisms which might have been present in the blood. After the treatment with penicillin was started there was a dramatic improvement in the child's condition. The highest temperature recorded on March 10 was 100·4° F., and by March 12 the highest reading was 99·4° F.; after this there was no rise in temperature. Tenderness of the left femur could still be elicited for some days, but X-ray examination on March 19 revealed no abnormality, and the child was discharged from hospital.

Dr. Nelson said that in spite of the failure of attempts at culture from the blood, he felt convinced that this was a case of septicæmia with involvement of the lung and early bone infection aborted by penicillin. Before the introduction of penicillin, this type of case was associated with a high mortality rate, and even if the patient survived the initial phase, a bone infection with months of invalidism usually followed. A number of patients had been treated in the hospital whose blood had yielded microorganisms on culture, and whose bone infection had been aborted by the use of penicillin.

Cerebro-Spinal Rhinorrhœa.

Dr. Nelson finally showed a female patient, aged nine years, who had been admitted to hospital on July 31, 1944, from a country hospital. One month previously she had fallen off a bicycle and sustained a fracture through the right frontal bone. Her progress had been satisfactory until two days before her admission to the Royal Alexandra Hospital for Children, when she began to suffer from violent headache, pain in the back and pyrexia, and she had been vomiting frequently.

On her admission to hospital she was evidently suffering from meningitis; severe head retraction and rigidity of the neck, the presence of Kernig's sign, pyrexia and turbid cerebro-spinal fluid were the outstanding signs. Microscopic examination of the cerebro-spinal fluid revealed numerous pus cells and some Gram-positive cocci in pairs. The protein content was estimated at forty milligrammes *per centum*, and culture yielded a growth of pneumococci. Treatment was commenced with sulphadiazine and the intravenous administration of saline solution, with some improvement, and on the third day, as there was a recurrence of pyrexia, penicillin was given by intramuscular injection, 10,000 units being administered every three hours until a total of 1,000,000 units had been given. The elevated temperature subsided, the general condition improved, and in three weeks she was considered fit for discharge from hospital.

Later the child reported to the follow-up department, and appeared quite well. Her aunt mentioned casually that she seemed to have a "cold in the head" and was frequently

blowing her nose. Careful questioning revealed the fact that she had an intermittent rhinorrhœa, which might cease for one day but no longer, and this had been present while she was in hospital. In view of the danger of recurrent meningitis the child was readmitted to hospital for operation. This was performed on October 10 and was preceded by a short course of sulphadiazine treatment. A coronal incision was made and the skin flap was turned down over the forehead. The fracture line could be felt running down the frontal bone towards the frontal sinus. A bone flap was reflected with its hinge on the temporal bone, and an extradural approach was made to the anterior fossa by elevation of the frontal lobe with its dural covering. It was found that the fracture extended down to the cribriform plate, and in this region there was a hernia of cerebral substance through the torn dura. This area was freed, and the dura was oversewn. The bone and soft tissue flaps were replaced, and a small drain was left in the angle of the wound for forty-eight hours. Post-operative convalescence was uneventful, and the rhinorrhœa did not recur.

Dr. Nelson remarked that it was of interest that no one noticed the rhinorrhœa while the child was in hospital. In some of these cases the tear closed spontaneously, but after two months it was considered that this was unlikely, and the operative findings confirmed this opinion. The simplest method of closing a fistula of this nature was by an extradural operation, even if it meant the raising of both frontal lobes as advocated by Adson; but if this method was impracticable owing to inaccessibility of the fistula, the dura had to be opened and repaired from within.

Extraarticular Fusion of the Hip Joint following Cured Tuberculous Arthritis.

DR. G. KEITH SMITH showed a male patient, aged fourteen years, who had been first admitted to hospital in September, 1936, with a history of having had a sore right hip for eight months and of limping when he walked. X-ray examination revealed that the right femur was slightly more transradiant than the left, but apart from this no definite change could be seen. Later X-ray appearances suggested early Perthes's disease. In 1939 an X-ray examination revealed an area of erosion in the acetabulum, which was increasing in size; the edges were becoming more indistinct and the cartilage was thinner than before. The X-ray appearances suggested a tuberculous infection.

Dr. Smith said that the child was treated first on a frame and later with plaster spicas. In 1941 X-ray examinations showed the lesion to be stationary. It was decided to perform an operation for fusion of the hip joint; this was carried out in December, 1943. A thin incision was made over the lateral aspect of the right hip joint. The femur was approached and an instrument was hammered through the femur below the head. A graft five inches long was taken from the tibia and the graft was inserted into the prepared femoral gap and hammered in. The wounds were closed and plaster casts were applied. X-ray examination four months later showed the graft to be uniting. The patient was discharged from hospital in April, 1944.

Dr. Smith remarked that arthrodesis of the hip joint in cases of cured tuberculous arthritis was undertaken in order to prevent the development of crippling flexion-adduction deformity. It had long been recognized that appliances in the convalescent and ambulatory stages of the disease had had little effect in the prevention of distortion. The operation described by Brittain had been carried out in the case under discussion; it involved an osteotomy at a suitable level and the insertion of a tibial graft under radiological control. Dr. Smith showed a series of X-ray films, illustrating the various stages in the development of new bone from the time of the placement of the graft. He said that the advantage of this operation was the mechanical stability obtained by a buttress of bone below the joint. The osteotomy allowed some correction, if necessary, in the weight-bearing alignment, the medial displacement of the lower fragment enhancing the stability.

Chronic Empyema with Decortication.

DR. H. G. HUMPHRIES showed a female patient, aged five years, who had been first admitted to hospital in December, 1940. The patient had had pertussis for three weeks; then bronchopneumonia developed, and in March, 1941, there were signs of fluid in the right side of the chest. Aspiration was performed and thick pus was obtained. A thoracotomy was then carried out. Drainage from the empyema wound continued, and three months later the wound was reopened and a drainage tube was inserted. After removal of the tube the sinus continued to discharge material. In June, 1944, it was decided to decorticate the lung. The first operation was performed on June 6, 1944, and consisted in decortication of the lower and middle lobes of the right lung. On October 6 decortication of the upper part of the right lung was performed. By January, 1945, the sinus was only one and a half inches deep, and it was decided to treat the condition with penicillin applied locally. At the time of the meeting the sinus was healed. X-ray examination on February 15, 1945, revealed on the right side operative collapse with gross pleural thickening, but no cavity, and no definite evidence of fluid. The lung was about 50% aerated.

Epispadias with Ureteral Implantation into the Bowel.

Dr. Humphries next showed a male child, aged three years, who had pronounced epispadias, almost *ectopia vesicæ*. Operation was performed on October 11, 1944. A right paramedian incision was made and the right ureter was transplanted into the sigmoid colon. The condition after operation was satisfactory, and the child passed urine *per rectum*. A similar operation was performed on the left side three months later. The child progressed well, and at the time of the meeting was passing urine *per rectum*.

Osteomyelitis.

DR. C. H. WESLEY showed a female patient, aged ten years, who had been admitted to hospital on March 26, 1945. She had complained of pain in the left thigh five days previously, and was said to have had a fall; she was delirious next day. The pain became increasingly severe and the hip became red and swollen.

On examination, the patient was flushed and obviously ill. Her temperature was 103° F. Redness and swelling were present, extending from the left iliac crest down to the knee, chiefly in the region of the hip joint. The left thigh was flexed and slightly abducted. Penicillin treatment was commenced, three cubic centimetres being given every three hours, and the child was also given sulphadiazine. Her general condition improved, but that of the thigh remained the same. She was nursed in extension. An X-ray examination on April 3 revealed early periostitis on both sides of the femur. At operation on April 13 an incision was made laterally over the upper part of the left thigh; a drill hole was made in the femur. A second incision was made at the lower end of the thigh. Drainage tubes were inserted for the local application of penicillin. Turbid fluid was found in the upper and lower incisions. Dr. Wesley said that the child was still being given penicillin; she had had 3,000,000 units up to the time of the meeting.

Dr. Wesley also showed a female patient, aged ten years, who was suffering from acute osteomyelitis of the ilium. She had been admitted to hospital on February 5, 1944, having been ill for five days. Her illness commenced with boils on the arms, legs and buttocks; she then complained of pain in the right thigh and leg, and was feverish.

On examination, the child was seen to be severely ill and slightly cyanosed. The right thigh was straight, and the hips were internally roated. Slight limitation of movement in all directions was observed. No abnormality was detected in the other systems. The child was given a blood transfusion and sulphathiazole. X-ray films of the hip joint and sacro-iliac joint revealed no abnormality. On February 12 an incision was made over the lateral side of the femur; no pus was found. *Staphylococcus aureus* was grown in culture from blood collected on February 5. X-ray examination on March 8 revealed a fairly large area of osteomyelitis just above the acetabulum, with septic arthritis; a fair-sized sequestrum was present near the upper part of the acetabulum.

The child's general condition improved. At a further operation on May 17 a sequestrum was removed from the acetabulum. The child's condition gradually improved, and she was nursed with the limb in fixed extension. An X-ray examination in September, 1944, revealed general improvement in the lesion, but considerable bone disturbance was still present and numerous sequestra were observed. Further operation was undertaken and the sequestra were removed. The child was given penicillin intramuscularly and also locally into the wound. Her general condition and also that of the wound improved rapidly. Two and a half million units of penicillin were given intramuscularly. X-ray examination in February, 1945, revealed no evidence of fresh foci or increased activity, and the cavity above the acetabulum was filling in.

Fractured Skull with Meningitis.

Dr. Wesley finally showed a female patient, aged fifteen months, who had been admitted to hospital on February 26, 1945. On that day she had fallen down seventeen steps and

hit her head on concrete. She did not become unconscious. She had vomited once, and had been drowsy since the accident.

On examination, the patient was seen to be pale and shocked; she was conscious, and her pulse rate was very rapid. A bruise was present over the right frontal area. The pupils were equal and reacted to light and accommodation. No paresis was present, the knee jerks were active and the plantar response was flexor in type. X-ray examination revealed a linear fracture passing downwards from the region of the anterior fontanelle.

On February 28 the child's general condition had improved, but weakness of the right arm and leg had developed. Her colour was poor. The following day her temperature was 103° F. A lumbar puncture was performed, and the cerebrospinal fluid was clear. A further lumbar puncture on March 3 yielded fluid containing 65 cells per cubic millimetre, 100% being polymorphonuclear cells. The chemical composition of the fluid was normal. *Staphylococcus aureus* was grown on culture.

The patient was treated with penicillin, two cubic centimetres intramuscularly every three hours, and sulphadiazine by mouth. The swelling on the left side of the forehead became pronounced, but the child's condition continued to improve. By March 10 she was moving her right arm and leg freely. The cerebro-spinal fluid still continued to contain an increased number of cells, and to yield *Staphylococcus aureus* on culture, till April 6; on that day there were only ten leucocytes per cubic millimetre, and no growth of micro-organisms was obtained on culture. Dr. Wesley said that the child was well, but was still being kept under observation.

(To be continued.)

Correspondence.

INJURIES BY UNKNOWN AGENTS TO BATHERS IN NORTH QUEENSLAND.

SIR: In your issue of April 21, 1945, Dr. H. Flecker took up a challenge to produce examples of the Portuguese man-o'-war or bluebottle, *Physalia*, from Great Barrier Reef waters. His claim, I am pleased to say, has now been substantiated, for I have just received from him two small examples collected alive on May 18 last at Green Island, a coral cay about sixteen miles east of Cairns. The specimens were part of a large number which were being driven onto the beach by the incoming tide.

This new evidence is most useful to me, and proves that the well-known float-supported marine stinger does invade at least one area of the more or less closed channel between the off-shore reefs and the coast of Queensland. As previously stated, I have a wide experience of the tropical Queensland waters during spring, summer and winter seasons, and could not establish that *Physalia* occurred in the area. This record near Cairns may possibly be accounted for by the proximity of a big break in the Barrier Reef (Trinity Opening) and the nearness of the main coral reef to the coast in that vicinity. Further to the south the open ocean beyond the Barrier Reef is as much as eighty to ninety miles east of the mainland coast, and of this distance the maze of compact coral beds takes up from forty to forty-five miles. I have specially mentioned these details because they describe a section of the Queensland waters with which I am particularly familiar—that is, between Mackay and Bowen. My most recent inquiries and observations were made there during eight or nine long launch cruises through the Cumberland and Whitsunday groups and to the Barrier Reef beyond. During them no information whatsoever on *Physalia* was elicited. In view of this I think that Dr. Flecker may concede me the opinion that the recorded deaths from marine stinging organisms in such places as Townsville and Pioneer Bay to the south of Bowen, are almost certainly due to an underwater swimming medusa (jellyfish) of the dread Carybdeid or sea wasp type. If *Physalia* had been the cause, it would certainly have been recognized by either the victims or witnesses of the tragedies. That a sea wasp, comparable to the lethal type responsible for the death of a youth at Darwin, does occur in Queensland waters has been established. A medical man in the services (Captain R. Southcott) has shown me drawings he made of a medusa collected in the sea near Cairns which bore the unmistakable characters of a sea wasp, of the same species or closely related to the specimen from Darwin preserved in the School of Tropical Medicine, University of Sydney.

Again I reiterate that the weals from sea wasp stings are of such an excoriated appearance that no reasonable comparison could be drawn between them and the non-lasting weals caused by *Physalia*. There is plenty of evidence of deeply excoriated weals suffered by bathers along the Queensland coast, but unfortunately these have not yet been linked with the causal organism which, I am convinced, is medusoid and sea wasp in character. It is hard to believe that *Physalia* is capable of killing a perfectly healthy person, as Dr. Flecker's letter in the issue of January 27 last appears to indicate. I would like to have some more details of the history of these cases and be convinced that the victims actually described the unmistakable blue float of *Physalia* as the main feature of the causal organism.

Dr. Flecker sums up his findings of the evidence to date in a list of five causal organisms—known and unknown (issue of April 21, 1945). This I wish to revise and modify, and submit the following:

(*a*) *Physalia* (Portuguese man-o'-war or bluebottle)—producing lash-like weals, more or less local symptoms, and lesions rarely lasting more than a day.

(*b*) Carybdeid medusa (sea wasp), responsible for the death of a ten-year-old boy at Darwin in 1939. Specimen in School of Tropical Medicine, Sydney. (Identified by F. A. McNeill.)

(*c*) Carybdeid medusa (sea wasp), either the same or closely related to the Darwin type and responsible for a number of deaths in Queensland waters. Stings associated with all deeply excoriated lesions lasting from six to eight days.

(*d*) Unknown organism producing insignificant and relatively unimportant local lesions and symptoms, but severe general symptoms. Commonly met with in at least the tropical Queensland waters around Cairns.

Yours, etc.,

F. A. McNEILL.

The Australian Museum,
Department of Invertebrates,
Sydney.
June 12, 1945.

TORSION OF THE FULL-TERM PREGNANT UTERUS.

SIR: The following is the report of a case of torsion through 180° of a full-term pregnant uterus. Such a case is so rare that it is not mentioned in most obstetrical text-books, and only one partly similar case has been reported in Australia (Shedden Adams), and only four similar cases have been reported in the literature during this century. Several cases of torsion in the early months of pregnancy have been reported, and especially when associated with a fibroid uterus, but a torsion through a full 180° at full term, without any associated abnormality as a cause, is an extreme rarity.

The patient, aged twenty-seven years, had had two previous normal confinements, and had an amputation of the cervix two years previously for leucorrhœa, resulting from a lacerated cervix. She was admitted to the King Edward Hospital for Women, Perth, with ruptured membranes, no contractions, vertex presenting, but floating high above the brim, and urine showed a heavy cloud of albumin.

Contractions started soon after admission, but six hours later the presenting part was still high, and meconium was present, and a funic souffle heard. The os admitted one finger, and an unusual band was felt anteriorly in the bladder region.

Two hours later the vaginal examination was the same, and, as the patient's general condition was deteriorating, I decided to perform a low segment Cæsarean section under cyclopropane anæsthesia, given by the honorary anæsthetist (Dr. Peacock). A right lower paramedium incision showed an extremely œdematous right Fallopian tube, and œdematous broad ligament lying over the site of the lower segment, with the ostium of the right Fallopian tube touching the left side of the abdominal wall.

Being unable to define the usual loose peritoneum indicating the lower uterine segment, I had to perform the classical incision over the side of a greatly thinned out sacculation of the uterus—so thin that, in making the incision, there was practically no hæmorrhage.

The baby was removed in the presence of much meconium, and is still alive. After suturing the incision, the cause of

the abnormality was then found to be a torsion through 180°—the actual site of the torsion being the lower uterine segment. There were no pathological tumours or adhesions in the abdomen, so, by rotating the uterus clockwise through 180°, the greatly œdematous right tube was now in its right position, and the uterine incision, which was in the midline anteriorly, was now in the midline posteriorly, and completely out of view.

Apart from the fact that during the twelve hours immediately following section there was only two drachms of urine in the bladder, her convalescence and that of the baby were moderately uneventful.

Incidentally, of the four similar cases reported in other parts of the world, two were discovered at post-mortem examination, the third died three days after Cæsarean section, and the fourth lived after Cæsarean section.

Yours, etc.,

ROLAND NATTRASS, D.G.O., M.R.C.O.G.

Leith House,
220, Saint George's Terrace,
Perth.
June 17, 1945.

DEAF AIDS.

SIR: The letter concerning deaf aids by "Interested" in your issue of June 16 instant is most pertinent.

Until comparatively recently, deaf aids magnified all sounds, with the result that loud accidental unwanted sounds, such as tapping the receiver *et cetera*, were amplified to a degree detrimental to the cochlea.

The modern valve-amplified deaf aid can be made with automatic volume control, just like the modern wireless receiver, so that no sounds over a certain volume come through. This improvement has made deaf aids useful and safe for the very deaf. Further, a greater number of cases of nerve deafness find them useful and all cases are glad to have excessive noise eliminated. It can therefore be anticipated that demands for deaf aids will greatly increase, and a decrease in prices should be expected.

Until manufacture of these articles is undertaken in Australia, ordering them by the prescription of an oto-rhino-laryngologist is out of the question, so meanwhile it is most important that patients be allowed to have instruments on trial or hire until they can estimate their value in the conditions where they would be used.

I am disappointed that an almost world-wide organization which is now pushing these instruments in Australia overlooked this essential point. Possibly the difficulty of replacing valves in wartime has something to do with the present position, which is, that patient after patient complains of firms refusing to lend or hire, even if the purchase sum be deposited meanwhile with a reputable person such as a bank manager.

It was, therefore, with pleasure that, on opening my mail this morning, I found a letter from a firm selling a very reliable make of deaf aid informing me that it had just lent two of my patients instruments for home trial.

I will have pleasure in forwarding the name of this firm to "Interested" or any other medical man who may wish to know it.

Yours, etc.,

A. B. K. WATKINS.

17, Bolton Street,
Newcastle,
New South Wales.
June 15, 1945.

RHEUMATISM.

SIR: I notice in the most recent edition of the *British Medical Journal* an article by Sir Adolphe Abrahams, in which he states that a scheme is under way in England to provide for the specialized post-graduate training of medical men in the recognition and treatment of the rheumatic diseases. It would appear that the British Government has decided to include in its medical benefits these diseases, and the Empire Rheumatism Council, to implement this step, advocates a course to enable practitioners to familiarize themselves with the multitudinous aspects of rheumatism.

These are procedures which I have recommended for many years, and if they have been found to be necessary in England, they are none the less so, proportionately, in Australia. In just on twenty years' exclusive practice in arthritic and psychological problems, I have been impressed repeatedly by three situations. Firstly, faulty diagnosis; secondly, inadequate and inappropriate treatment; thirdly, the complaint by hundreds of patients, that so far as they knew, there were no clinics and no specialists in Australia.

Only this morning I saw a man who has been under treatment for eight years with five medical men and three unqualified practitioners, without benefit, for a so-called rheumatoid arthritis of the fingers. Nobody in that time apparently suggested taking even a skiagram.

Rheumatology is such a complex branch of medicine that each case requires the fullest investigation from every angle, and as far as I am aware, the facilities for either this, or adequate treatment, are not available in any of the hospitals. There are also many patients who are unable to pay the fees in private practice for what must necessarily be a lengthy and expensive treatment, and surely with all this talk of post-war reconstruction, a department should be instituted in each of our hospitals to handle these diseases. This should be run in collaboration with the physician, the orthopædic surgeon and the neurosurgeon and have full benefit of radiological and pathological advice. Then we might make some headway, and not merit the oft-heard reproach: "Nothing can be done for arthritis."

Yours, etc.,

E. HASLETT FRAZER.

231, Macquarie Street,
Sydney,
June 22, 1945.

LIGATURE OF THE POPLITEAL ARTERY.

SIR: I should like to congratulate Colonel Yeates on an excellent result in his case of ligature of the popliteal artery for gunshot wound (THE MEDICAL JOURNAL OF AUSTRALIA, June 23, 1945, page 637). In recording details of the case he has performed a service, since satisfactory statistics of the results of popliteal ligature are much needed, and these can only be obtained from a series of carefully recorded cases such as his.

He suggests that the high site of the ligature near the opening in the *adductor magnus* and above the origin of the articular branches of the artery was probably the reason for the peripheral circulation remaining adequate. While agreeing with this observation, I would also suggest that the fact that the damage to the artery was caused by the point of a bullet and not by a shell fragment, was probably also important, since there had been but little damage to the surroundings of the artery in the popliteal fossa. Colonel Yeates comments on the minor extent of the muscle damage and the relatively clean track of the bullet in the case he records. When a jagged shell fragment damages the popliteal artery, it usually also causes widespread damage of the surrounding muscles and the muscular branches of the artery which are important as collateral vessels. It thereby severely jeopardizes the peripheral circulation in a way which a clean bullet wound does not do.

Colonel Yeates asks whether there is a crucial point in the popliteal artery analogous to the "critical point" of Sudeck in the inferior mesenteric artery. The high site above the origin of the articular branches may well prove to be the safest part of the course of the artery in which to apply a ligature, but I suggest that the question of the nature of the missile which has caused the wound, and therefore of the extent of the damage to surrounding muscle, may equally affect the issue. I would add to his plea, therefore, that in the compilation of statistics for gangrene following popliteal ligation the exact anatomical level of the lesion be stated, one for recording also the type of missile causing the arterial injury and the extent of the associated damage in the popliteal fossa, particularly to the muscular contents.

Yours, etc.,

LAMBERT ROGERS,
Surgical Consultant, British Pacific Fleet.

June 25, 1945.

VENEREAL DISEASE?

SIR: Dr. C. P. Harrison's letter on veneral diseases published in your issue of the 16th instant expresses exactly the sentiments that I have volunteered to both lay and medical authorities in South Australia.

My recommendations were mainly directed towards encouraging suspects and sufferers to apply for and con-

tinue treatment. It had generally been conceded that, for this purpose, investigation should be more confidential, or even secret, at the so-called venereal clinics.

I pointed out, as does Dr. Harrison, that venereal disease is not an entity, but a collection of various disorders that have nothing in common with each other apart from the usual mode of origin: to continue treating them as one, we were helping to perpetuate a mediæval fallacy that the social disorder was more important to us than the medical.

Syphilis, for instance, has much less claim to be allied to gonorrhœa than to measles, and that long before the primary genital lesion has disappeared it has become a systemic disease, such as tuberculosis. A person with a tuberculous lesion on or in his genitals would never be wittingly kept in a veneral disease clinic. Gonorrhœa, on the other hand, is a genito-urinary infection, and has as much claim to be treated in a urological clinic as a B.C.C. pyelitis.

The fact that non-specific cases of prostato-vesiculitis are referred to the venereal disease clinic for treatment, because there is no separate satisfactory organization for their treatment, and because treatment is similar to gonococcal infections of the same organs, is a precedent for treating all genito-urinary infections in the same clinic. In this way the victims of infections that had been contracted in a socially repulsive manner would not feel that they were being branded, and the innocent prostatitis cases would be relieved of the same (in their case) unfair stigma.

The late Frank Kidd stated that a venereologist should be primarily a urologist. Only one with special knowledge of urology will sift out the many cases of tuberculous kidney, bladder diverticula, strictures, not to mention many other non-venereal conditions that come to a venereal disease clinic.

Before we can educate the public to be treated scientifically, the medical profession itself must be educated to the fact that venereal disease to us is not a social entity but a variety of medical problems.

Yet both medical and political authorities have disagreed with me. One quite independent view, that of a police inspector, all the more valuable as that of a layman, advocated a polyclinic, from which the name "venereal" should be omitted.

Yours, etc.,

175, North Terrace,
Adelaide,
June 20, 1945.

W. JOHN CLOSE.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

FELLOWSHIPS IN THE FACULTY OF MEDICINE.

THE following fellowships are available in the Faculty of Medicine of the University of Sydney. Further information and application forms may be obtained from the Dean of the Faculty.

Walter and Eliza Hall Travelling Fellowship. Tenable for two years abroad and one year at the University of Sydney in any branch of medicine. Value: £300 to £500 *per annum*.

Liston Wilson Research Fellowship in neurological subjects. Value: £400 *per annum*.

Reginald Lake Research Fellowship in Pathology. Value: £400 *per annum*.

Gordon Craig Fellowship in Urology—tenable at the University and Royal Prince Alfred Hospital (residence). Value: £400 *per annum*.

Marion Clare Reddall Fellowship in General Medical Science. Value: £400 *per annum*.

Anderson Stuart Fellowship in General Medical Science. Value: £400 *per annum*.

Sister Sanders Fellowship in Pædiatrics (residence at Royal Alexandra Hospital for Children). Value: £400 *per annum*.

Wyeth Fellowships in Obstetrics (resident)—one tenable at Royal Hospital for Women and one at Women's Hospital, Crown Street, under the control of the Department of Obstetrics. Value: £400 *per annum*.

Nuffield Travelling Fellowship in any branch of medicine. Tenable at Oxford for three years with three years at the University of Sydney. Value: £400 to £600 *per annum*. These are only allocated at irregular intervals and very few are available.

Research grants are available for research anatomy under the control of the Department of Anatomy.

The National Health and Medical Research Council also grants junior research fellowships at £500 *per annum* to suitable applicants with research experience for work in university departments, the conditions being laid down by the council.

Naval, Military and Air Force.

DECORATIONS.

THE following honours and awards have been announced in the *Commonwealth of Australia Gazette*, Number 54, of March 15, 1945.

Companion of the Most Excellent Order of the British Empire.—Colonel F. H. Beare, E.D., Colonel J. C. Belisario, O.B.E., Colonel A. H. Green, Colonel J. Leah.

Officer of the Most Excellent Order of the British Empire.—Colonel R. H. MacDonald, Colonel A. J. Murray, Major P. A. Tomlinson.

Military Cross.—Captain H. Glynn-Connolly.

Mentioned in Dispatches.—Brigadier H. G. Furnell, C.B.E., D.S.O., Colonel B. S. Hanson, D.S.O., O.B.E., Colonel J. E. Gillespie, Lieutenant-Colonel P. W. Hopkins, M.C., Lieutenant-Colonel G. B. D. Hall, Lieutenant-Colonel E. W. Kyle, Lieutenant-Colonel W. E. E. Langford, Lieutenant-Colonel L. M. Outridge, Lieutenant-Colonel C. A. M. Renou, Lieutenant-Colonel C. M. Sangster, Lieutenant-Colonel J. E. Sewell, Lieutenant-Colonel J. H. Thorp, Lieutenant-Colonel S. W. Williams, Lieutenant-Colonel R. F. A. Becke, Major J. F. C. C. Cobley, Lieutenant-Colonel S. J. Douglas, Major C. J. Gibson, Major M. R. Gold, Major G. C. Love, Major S. L. Mainland, O.B.E., Lieutenant-Colonel G. N. Morris, Lieutenant-Colonel R. G. Quinn, Captain D. S. Brandt, Major H. J. Edelman, Captain W. Gove, Major A. A. Merritt, Captain S. Rose, Major W. P. Ryan.

CASUALTIES.

ACCORDING to the casualty list received on June 27, 1945, Captain N. G. Hoddle, A.A.M.C., Pymble, New South Wales, has now been removed from the "seriously ill" list.

According to the casualty list received on June 28, 1945, Captain M. Mayrhofer, A.A.M.C., Merredin, Western Australia, who was previously reported to be a prisoner of war, is now reported as "not prisoner of war".

Obituary.

ALLEN ERNEST RAMSEY.

WE regret to announce the death of Dr. Allen Ernest Ramsey, which occurred on June 25, 1945, at Cremorne, New South Wales.

LUDWIG AUGUST KORTUM.

WE regret to announce the death of Dr. Ludwig August Kortum, which occurred on June 28, 1945, at Auburn, New South Wales.

MATTHEW KASNER MOSS.

WE regret to announce the death of Dr. Matthew Kasner Moss, which occurred on June 28, 1945, at Perth.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Smith, Warren James, provisional registration, 1945 (Univ. Sydney), 14, Chelmsford Avenue, Croydon.

Chancellor, Alan Harold Bennet, provisional registration 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association as from July 1, 1945:

Burkitt, Arthur Robert, M.B., B.S., 1944 (Univ. Sydney), 108, Albert Road, Homebush.

Cassidy, Desmond John, M.B., B.S., 1944 (Univ. Sydney), 112, Lang Road, Centennial Park.

Child, Margaret Eileen, M.B., Ch.B., 1930 (Univ. Glasgow), Nincoola, Guyra.

Cleland, Kenneth Wollaston, provisional registration, 1945 (Univ. Sydney), Goulburn District Hospital, Goulburn.

Fogarty, Diarmuid, M.B., B.S., 1941 (Univ. Sydney), 137, George Street, Bathurst.

Freeman, Zelman Samuel, M.B., B.S., 1944 (Univ. Sydney), Sydney Hospital, Sydney.

Green, Robert Musgrave, provisional registration, 1945 (Univ. Sydney), Sydney Hospital, Sydney.

Holley, William Chalfont, M.B., B.S., 1938 (Univ. Sydney), 12, Lancaster Road, Rose Bay.

Howe, René Edgar, provisional registration, 1945 (Univ. Sydney), Royal North Shore Hospital, St. Leonards.

Kerkenezov, Nicholas, M.B., 1943 (Univ. Sydney), Pennant Hills Road, Carlingford.

Lofberg, Jack Valdemar, provisional registration, 1945 (Univ. Sydney), 30, Albyn Street, Bexley.

Moore, David Robert, provisional registration, 1945 (Univ. Sydney), Balmain and District Hospital, Balmain.

Parker, Anthony Owen, M.B., B.S., 1942 (Univ. Sydney), 738, New South Head Road, Rose Bay.

Silvester, George Raymond, provisional registration, 1945 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Sinclair-Smith, Bruce Cooper, M.B., B.S., 1943 (Univ. Sydney), 13, Wentworth Road, Vaucluse.

Friedlander, Eric Martin Caesar (registered as Erich Martin Caesar Friedlaender), M.D., 1908 (Univ. Giessen), recommended and approved for registration in terms of Section 17 (2) of the *Medical Practitioners Act*, 1938, 141, Macquarie Street, Sydney.

Medical Appointments.

Dr. Glen Howard Burnell, Dr. Brian Herbert Swift and Dr. Eugene McLaughlin have been appointed by the Board of Management of the Royal Adelaide Hospital to be Honorary Surgeon, Honorary Gynæcologist and Honorary Consultant Physician respectively to the Sterility Clinic, Royal Adelaide Hospital.

Dr. Jack Sidney Anderson has been appointed as Out-Patients' Registrar of the Royal Adelaide Hospital.

Dr. Joseph Ruskin Cornish has been appointed Temporary Assistant Medical Superintendent of the Royal Adelaide Hospital.

Dr. Lewis Wibmer Jeffries, Dr. Alfred Burgess Russell and Dr. Keith McEwin have been appointed members of the Nurses' Board of South Australia under the provisions of the *Nurses Registration Act*, 1920-1938.

Books Received.

"Clinical Atlas of Blood Diseases", by A. Piney, M.D., M.R.C.P., and Stanley Wyard, M.D., F.R.C.P.; Sixth Edition; 1945. London: J. and A. Churchill Limited. 8" × 5¼", pp. 146, with 48 illustrations, 45 in colour. Price: 16s.

"Lettsom: His Life, Times, Friends and Descendants", by James Johnston Abraham; 1933. London: William Heinemann Medical Books Limited. 9¾" × 7½", pp. 518, with many illustrations. Price (Australian): 23s. 9d.

Diary for the Month.

JULY 10.—Tasmanian Branch, B.M.A.: Ordinary Meeting.
JULY 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 10.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JULY 13.—Queensland Branch, B.M.A.: Council Meeting.
JULY 16.—Victorian Branch, B.M.A.: Hospital Subcommittee.
JULY 16.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
JULY 17.—Victorian Branch, B.M.A.: Organization Subcommittee.
JULY 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 18.—Western Australian Branch, B.M.A.: General Meeting.
JULY 19.—South Australian Branch, B.M.A.: Council Meeting.
JULY 19.—Victorian Branch, B.M.A.: Executive Meeting.
JULY 19.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 24.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 25.—Victorian Branch, B.M.A.: Council Meeting.
JULY 26.—South Australian Branch, B.M.A.: Scientific Meeting.
JULY 26.—New South Wales Branch, B.M.A.: Branch Meeting.
JULY 27.—Queensland Branch, B.M.A.: Council Meeting.
AUG. 1.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 1.—Western Australian Branch, B.M.A.: Council Meeting.
AUG. 2.—South Australian Branch, B.M.A.: Council Meeting.
AUG. 3.—Queensland Branch, B.M.A.: Branch Meeting.
AUG. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

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